

A Dissertation on

**“COMPARATIVE STUDY BETWEEN POST OPERATIVE EARLY
VERSUS LATE ENTERAL NUTRITION IN PATIENTS UNDERGOING
ELECTIVE LAPAROTOMIES IN A TERTIARY CARE HOSPITAL “.**

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Branch – I



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BONAFIDE CERTIFICATE

Certified that this dissertation is the bonafide work of

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DECLARATION

I , certainly declare that this dissertation titled, “COMPARATIVE STUDY BETWEEN POST OPERATIVE EARLY VERSUS LATE ENTERAL NUTRITION IN PATIENTS UNDERGOING ELECTIVE LAPAROTOMIES IN A TERTIARY CARE HOSPITAL ” ,represent a genuine work of mine . The contribution of any supervisors to the research are consistant with normal supervisory practice, and are acknowledged.

I , also affirm that this bonafide work or part of this work was not submitted by me or any others for any award , degree or diploma to any other university board , neither in India or abroad . This is submitted to The Tamil Nadu Dr.MGR Medical University, Chennai in partial fulfilment of the rules and regulation for the award of Master of Surgery Degree Branch 1 (General Surgery) .

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ABSTRACT

COMPARATIVE STUDY BETWEEN POST OPERATIVE EARLY VERSUS LATE ENTERAL NUTRITION IN PATIENTS UNDERGOING ELECTIVE LAPAROTOMIES IN A TERTIARY CARE HOSPITAL

INTRODUCTION

Post operative starvation is the most common wide spread practice after gastro Intestinal surgery. The rationale of nil by mouth and gastric decompression is to prevent post operative nausea and vomiting and protect the anastomosis allowing it time to heal before being stressed by food. But in actual practice deferral of post operative enteral nutrition may be harmful.

OBJECTIVES OF THE STUDY

To study the impact of early enteral feeding as compared to late enteral feeding post operatively in patients undergoing elective laparotomies

- 1.Return of bowel function.
- 2.Incidence of general complications
- 3.Incidence of surgery specific complications and mortality
- 4.Total duration of hospital stay

MATERIALS & METHODS

All patients undergoing elective laparotomies including upper gastrointestinal, hepatobiliary, colorectal and surgeries involving excessive bowel handling in the Department of General Surgery at Rajiv Gandhi Government General Hospital ,Chennai-600003 in the period of June 2013 to November 2013 are included in this study.

The study is a prospective study. Study volume 100 patients.

CONCLUSION

The final conclusions drawn from this study comparing early versus late enteral nutrition in the post operative patients undergoing elective laparotomies are as follows.

- Length of hospital stay, wound infection, general complications not direct consequence of surgery is significantly decreased in early enteral feeding group.
- No significant difference was noted with Anastomotic leak, paralytic ileus rate between two groups.
- Patients tolerance for oral feeding was better in late feeding.

INTRODUCTION

Post operative starvation is the most common wide spread practice after gastro Intestinal surgery. The rationale of nil by mouth and gastric decompression is to prevent post operative nausea and vomiting and protect the anastomosis allowing it time to heal before being stressed by food.

“Early feeding may enhance wound healing and crease anastomotic strength particularly in malnourished patients. Pre existing malnutrition is a major clinical problem in surgical patients. Nutritional depletion is an independent determinant of serious complications after major gastrointestinal surgery Early nutritional support was associated with significant reduction in post operative complications, a reduction that was independent of pre operative nutritional status.”

“The benefits of post operative Enteral feeding in normally nourished surgical patients indicate that it is reduced nutritional intake that predisposes to develop complications, including deficits in muscle function and fatigue. Early post operative Enteral nutrition either afforded

no advantage over standard care or seemed to have a deleterious effect".

“Early post operative Enteral nutrition may have a beneficial effect on function of intestinal barrier in respect of permeability, bacterial translocation and subsequent development of septic complications. Early post operative nutrition influences intestinal permeability".

OBJECTIVES OF THE STUDY

To study the impact of early enteral feeding as compared to late enteral feeding post operatively in patients undergoing elective laparotomies in the Department of General Surgery, Rajiv Gandhi Government General Hospital, Chennai-03, with respect to

1.Return of bowel function.

2.Incidence of general complications

3.Incidence of surgery specific complications and mortality

4.Total duration of hospital stay

THEORY ASPECT

Anatomy and Physiology of Stomach

“The stomach (Ventriculus or gaster) is situated between the lower end of the esophagus and the beginning of the small intestine. It lies in the epigastric, umbilical and left hypochondriac regions of the abdomen, and occupies a recess bounded by the upper abdominal viscera and completed in front and on the left side by the anterior abdominal wall and the diaphragm.”

Its **mean capacity** varies with age, being about 30 ml at birth, increasing gradually to about 1000 ml at puberty and commonly reaching to 1500 ml in the adult.

The opening by which the esophagus communicates with the stomach is the "**cardiac orifice**" and is situated on the left of the median plane, behind the 7th costal cartilage 2.5cms from its junction with the sternum and at the level of eleventh thoracic vertebra. It is placed about 10 cm (4 inches) from the anterior abdominal wall and is 40 cm (16 inches) from the incisor teeth.

The opening into the duodenum is the "**pyloric orifice**" and its position is usually indicated by a circular groove on the surface of the organ, termed the "**pyloric constriction**" which indicates the position of the pyloric sphincter. In the living subject, at operation, it can be identified by the **prepyloric vein of Mayo**, which runs vertically across its anterior surface. The pyloric orifice lies about 1-2 cm to the right of the median plane in the transpyloric plane passing along the ninth costal cartilages at the level of the lower border of the first lumbar vertebra.

The Stomach Wall:

“ It consists of four layers — mucosa, submucosa, muscularis mucosa and serosa[20].

Mucosa-- The mucous membrane is thick and its surface is smooth, soft and velvety. During the contracted state of the organ it is thrown into numerous or rugae which for the most part have a longitudinal direction, and are best marked towards the pyloric end of the stomach, and along the greater curvature. These folds are obliterated when the organ is

distended. All the secretory elements are within the mucosa.”

Submucosa — Is mainly made up of areolar connective tissue, blood and lymphatic plexus.**Muscularis mucosa** — Consists of thin layers of inner circular and outer longitudinal muscles.

Muscularis propria — Consist of three muscle layers the distribution of which varies according to the site.

1.Inner oblique muscle layer extends from the body of the stomach to the pyloric sphincter.

2.Middle circular layer is mainly in the body of the stomach to pyloric sphincter.

3.Outer longitudinal layer mainly extends along the lesser curvature.

Serosa - The serosa or visceral peritoneum, covers the entire surface of the organ excepting few regions.

The surface of the mucous membrane including gastric pits is covered with a single layer of secretory columnar epithelial cells, the surface mucous cells,

which liberate mucous from their apices on to the surface of the stomach. This acts as the lubricant and protects the gastric lining against its own secretions of acids and enzymes.

Gastric glands:

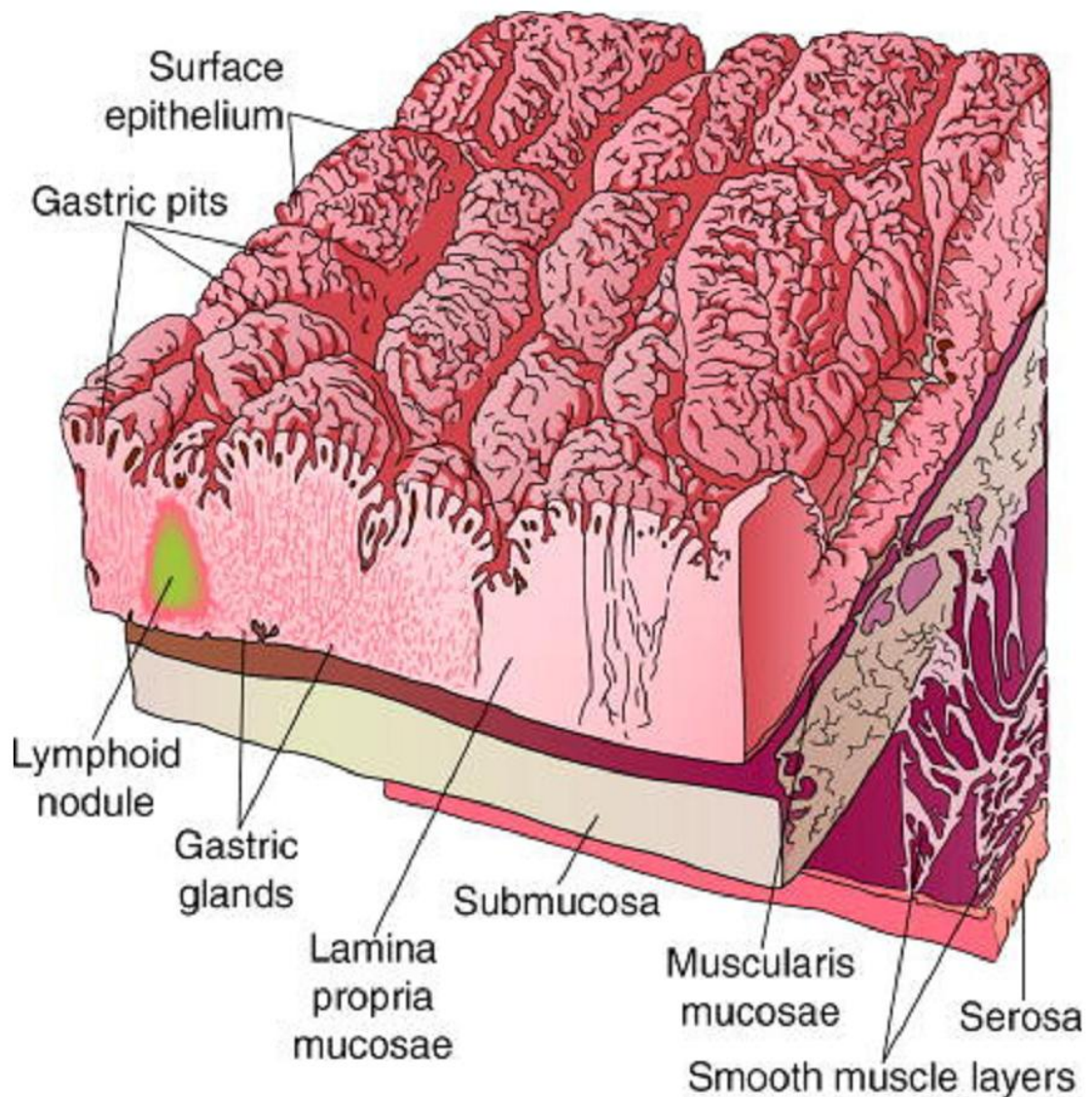
Cardiac glands: Mucous, endocrine, undifferentiated cells

Parietal cells secrete acid found in Fundus and body:

Oxyntic glands or **Chief** cells secrete — Pepsinogen, Intrinsic factor, Gastric enzymes.\

Antrum: Antral glands — Endocrine, mucous, parietal cells

G cells — Gastrin secreting cells



Blood supply:

Blood supply of the stomach is derived from the various branches of the celiac plexus[20]. The venous equivalent of left gastric artery is called as coronary vein. The gastric veins commence as straight vessels between the glands of the mucosa and these drain into submucosal veins. Larger veins accompany the corresponding

arteries to their ultimate drainage into splenic and superior mesenteric veins, while some pass directly to portal vein.

There are rich extramural and intramural collateral vessels. Mucosal blood flow is one of the key factors in defense against injury.

Lymphatic drainage:

Here also extensive intramural and extramural communications exist. As a consequence, malignancy spreads intramurally beyond the region of the origin.

Nerve supply:

Sympathetic:

Preganglionic fibres arising predominantly from T6 to T8. Sympathetic fibres subserve visceral sensation and pain.

Parasympathetic:

Parasympathetic innervation occurs by right and left vagus nerves. They form the distal esophageal plexus and give rise to right and left vagi, which pass through the esophageal hiatus of the diaphragm.

The left trunk is usually closely adherent to the anterior surfaces of the esophagus, whereas right trunk is often midway between the esophagus and the aorta. The left vagus supplies a hepatic branch passing to the right in the lesser omentum before innervating the liver and biliary tract. The remaining anterior vagal fibres parallel the left curvature of the stomach and branch into the anterior gastric wall.

Branch of anterior vagus:

1. Hepatic branches

2. Pyloric nerve of Mc Crae — inconstant nerve in pyloric region

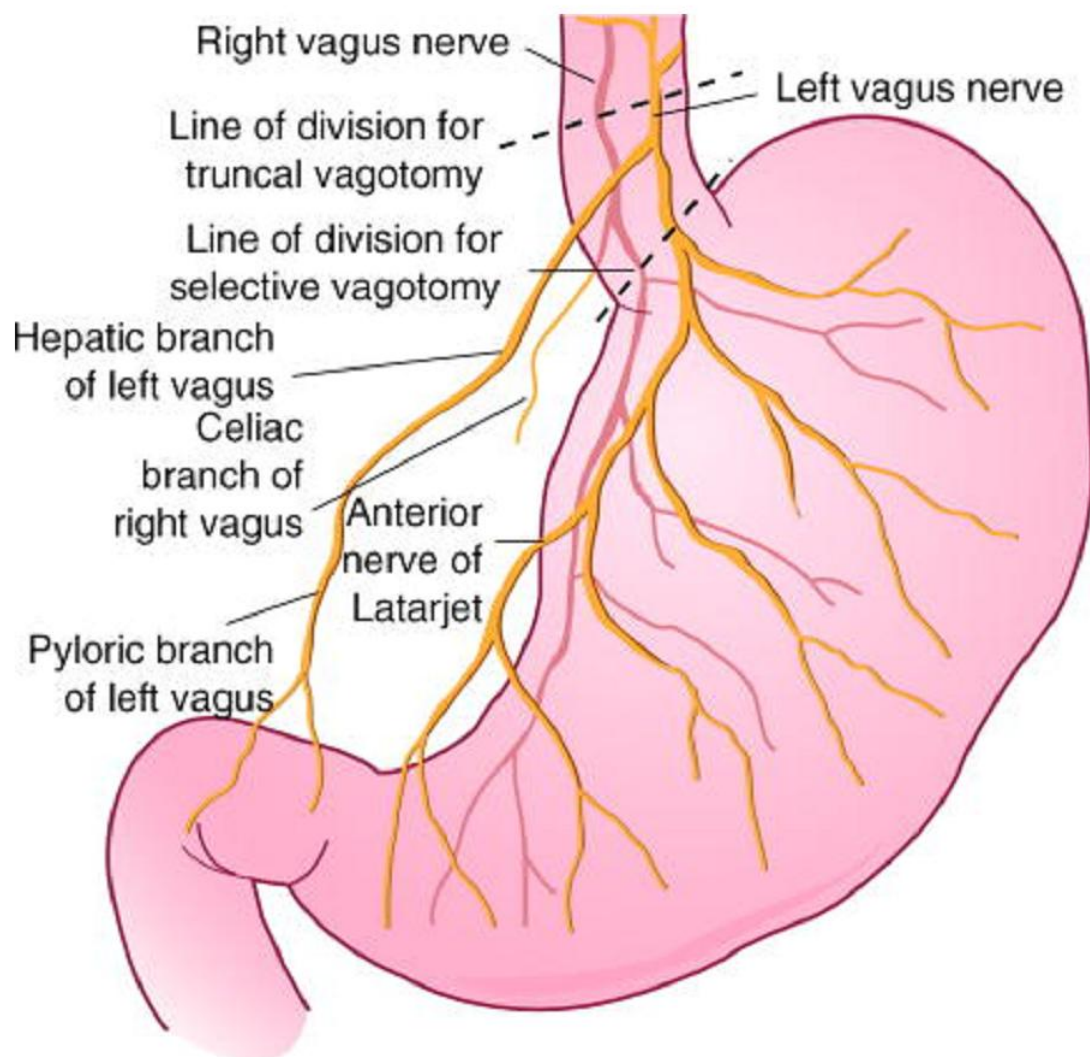
3. Anterior division / nerve of Latarget (preserved in HSV)

Nerve of Grassi (one of the branches of Latarget — held responsible for recurrence of ulcer)

The right or the posterior vagus supplies a coeliac branch to the coeliac plexus and the posterior gastric division, innervating the posterior gastric wall.

Branch of posterior vagus:

1. Posterior division / posterior nerve of Latarjet
2. Coeliac division of coeliac plexus



PERISTALSIS : “Peristalsis is a reflex response that is initiated when the gut wall is stretched by the contents of the lumen, and it occurs in all parts of the gastrointestinal tract from the esophagus to the rectum. The stretch initiates a circular contraction behind the stimulus and an area of relaxation in front of it. The wave of contraction then moves in an oral-to-caudal direction, propelling the contents of the lumen forward at rates that vary from 2 to 25 cm/s. Peristaltic activity can be increased or decreased by the autonomic input to the gut, but its occurrence is independent of the extrinsic innervation. Indeed, progression of the contents is not blocked by removal and resuture of a segment of intestine in its original position and is blocked only if the segment is reversed before it is sewn back into place. Peristalsis is an excellent example of the integrated activity of the enteric nervous system. It appears that local stretch releases serotonin, which activates sensory neurons that activate the myenteric plexus. Cholinergic neurons passing in a retrograde direction in this plexus activate neurons that release substance P and acetylcholine, causing smooth muscle contraction. At the same time, cholinergic neurons passing in an anterograde direction activate neurons that secrete NO, vasoactive intestinal polypeptide (VIP), and adenosine triphosphate (ATP), producing the relaxation ahead of the stimulus.”

SEGMENTATION & MIXING

When the meal is present, the enteric nervous system promotes a motility pattern that is related to peristalsis, but is designed to retard the movement of the intestinal contents along the length of the intestinal tract to provide time for digestion and absorption. This motility pattern is known as segmentation, and it provides for ample mixing of the intestinal contents (known as chyme) with the digestive juices.

BASIC ELECTRICAL ACTIVITY & REGULATION OF MOTILITY

Except in the esophagus and the proximal portion of the stomach, the smooth muscle of the gastrointestinal tract has spontaneous rhythmic fluctuations in membrane potential between about -65 and -45 mV. This basic electrical rhythm (BER) is initiated by the interstitial cells of Cajal, stellate mesenchymal pacemaker cells with smooth muscle-like features that send long multiply branched processes into the intestinal smooth muscle. In the stomach and the small intestine, these cells are located in the outer circular muscle layer near the myenteric plexus; in the colon, they are at the submucosal border of the circular muscle layer. In the stomach and small intestine, there is a descending gradient in pacemaker frequency, and as in the heart, the pacemaker with the highest frequency usually dominates.

MIGRATING MOTOR COMPLEX

During fasting between periods of digestion, the pattern of electrical and motor activity in gastrointestinal smooth muscle becomes modified so that cycles of motor activity migrate from the stomach to the distal ileum. Each cycle, or migrating motor complex (MMC), starts with a quiescent period (phase I), continues with a period of irregular electrical and mechanical activity (phase II), and ends with a burst of regular activity (phase III). The MMCs are initiated by motilin, migrate aborally at a rate of about 5 cm/min, and occur at intervals of approximately 90 min. Gastric secretion, bile flow, and pancreatic secretion increase during each MMC. They likely serve to clear the stomach and small intestine of luminal contents in preparation for the next meal. They are immediately stopped by ingestion of food (which suppresses motilin release via mechanisms that have not yet been elucidated), with a return to peristalsis and the other forms of BER and spike potentials.

GASTRIC MOTILITY & EMPTYING

“When food enters the stomach, the fundus and upper portion of the body relax and accommodate the food with little if any increase in pressure (receptive relaxation). Peristalsis then begins in the lower portion of the body, mixing and grinding the food and permitting small, semiliquid portions of it to pass through the pylorus and enter the duodenum. Receptive relaxation is vagally mediated and triggered by movement of the pharynx and esophagus. Peristaltic waves

controlled by the gastric BER begin soon thereafter and sweep toward the pylorus. The contraction of the distal stomach caused by each wave is sometimes called antral systole and can last up to 10 s. Waves occur three to four times per minute. In the regulation of gastric emptying, the antrum, pylorus, and upper duodenum apparently function as a unit. Contraction of the antrum is followed by sequential contraction of the pyloric region and the duodenum. In the antrum, partial contraction ahead of the advancing gastric contents prevents solid masses from entering the duodenum, and they are mixed and crushed instead. The more liquid gastric contents are squirted a bit at a time into the small intestine. Normally, regurgitation from the duodenum does not occur, because the contraction of the pyloric segment tends to persist slightly longer than that of the duodenum. The prevention of regurgitation may also be due to the stimulating action of cholecystokinin (CCK) and secretin on the pyloric sphincter.”

REGULATION OF GASTRIC MOTILITY & EMPTYING

The rate at which the stomach empties into the duodenum depends on the type of food ingested. Food rich in carbohydrate leaves the stomach in a few hours. Protein-rich food leaves more slowly, and emptying is slowest after a meal containing fat. The rate of emptying also depends on the osmotic pressure of the material entering the duodenum. Hyperosmolality of the duodenal contents is sensed by “duodenal osmoreceptors” that initiate a decrease in gastric

emptying which is probably neural in origin. Fats, carbohydrates, and acid in the duodenum inhibit gastric acid and pepsin secretion and gastric motility via neural and hormonal mechanisms. The hormone involved is probably peptide YY. CCK has also been implicated as an inhibitor of gastric emptying

VOMITING

Vomiting is an example of central regulation of gut motility functions. Vomiting starts with salivation and the sensation of nausea. Reverse peristalsis empties material from the upper part of the small intestine into the stomach. The glottis closes, preventing aspiration of vomitus into the trachea. The breath is held in mid inspiration. The muscles of the abdominal wall contract, and because the chest is held in a fixed position, the contraction increases intra-abdominal pressure. The lower esophageal sphincter and the esophagus relax, and the gastric contents are ejected. The “vomiting center” in the reticular formation of the medulla consists of various scattered groups of neurons in this region that control the different components of the vomiting act.

Irritation of the mucosa of the upper gastrointestinal tract is one trigger for vomiting. Impulses are relayed from the mucosa to the medulla over visceral afferent pathways in the sympathetic nerves and vagi. Other causes of vomiting can arise centrally. For example, afferents from the vestibular nuclei mediate the nausea and vomiting of motion sickness. Other afferents presumably reach the vomiting control areas from the diencephalon and limbic system, because

emetic responses to emotionally charged stimuli also occur. Thus, we speak of “nauseating smells” and “sickening sights.” Chemoreceptor cells in the medulla can also initiate vomiting when they are stimulated by certain circulating chemical agents. The chemoreceptor trigger zone in which these cells are located is in the area postrema, a V-shaped band of tissue on the lateral walls of the fourth ventricle near the obex. This structure is one of the circumventricular organs and is not protected by the blood– brain barrier. Lesions of the area postrema have little effect on the vomiting response to gastrointestinal irritation or motion sickness, but abolish the vomiting that follows injection of apomorphine and a number of other emetic drugs. Such lesions also decrease vomiting in uremia and radiation sickness, both of which may be associated with endogenous production of circulating emetic substances. Serotonin (5-HT) released from enterochromaffin cells in the small intestine appears to initiate impulses via 5-HT₃ receptors that trigger vomiting. In addition, there are dopamine D₂ receptors and 5-HT₃ receptors in the area postrema and adjacent nucleus of the solitary tract. 5-HT₃ antagonists such as ondansetron and D₂ antagonists such as chlorpromazine and haloperidol are effective antiemetic agents. Corticosteroids, cannabinoids, and benzodiazepines, alone or in combination with 5-HT₃ and D₂ antagonists, are also useful in treatment of the vomiting produced by chemotherapy. The mechanisms of action of corticosteroids and cannabinoids are unknown,

whereas the benzodiazepines probably reduce the anxiety associated with chemotherapy.

INTESTINAL MOTILITY

The MMCs that pass along the intestine at regular intervals in the fasting state and their replacement by peristaltic and other contractions controlled by the BER are described above. In the small intestine, there are an average of 12 BER cycles/min in the proximal jejunum, declining to 8/min in the distal ileum. There are three types of smooth muscle contractions: peristaltic waves, segmentation contractions, and tonic contractions. Peristalsis is described above. It propels the intestinal contents (chyme) toward the large intestines. Segmentation contractions, also described above, move the chyme to and fro and increase its exposure to the mucosal surface. These contractions are initiated by focal increases in Ca^{2+} influx with waves of increased Ca^{2+} concentration spreading

from each focus. Tonic contractions are relatively prolonged contractions that in effect isolate one segment of the intestine from another. Note that these last two types of contractions slow transit in the small intestine to the point that the transit time is actually longer in the fed than in the fasted state. This permits longer contact of the chyme with the enterocytes and fosters absorption

MOTILITY OF THE COLON

The ileum is linked to the colon by a structure known as the ileocecal valve, which restricts reflux of colonic contents, and particularly the large numbers of commensal bacteria, into the relatively sterile ileum. The portion of the ileum containing the ileocecal valve projects slightly into the cecum, so that increases in colonic pressure squeeze it shut, whereas increases in ileal pressure open it. It is normally closed. Each time a peristaltic wave reaches it, it opens briefly, permitting some of the ileal chyme to squirt into the cecum. When food leaves the stomach, the cecum relaxes and the passage of chyme through the ileocecal valve increases (gastroileal reflex). This is presumably a vagal reflex.

ENTERAL NUTRITION

“ The term EN is used to comprise all forms of nutritional support that imply the use of “dietary foods for special medical purposes” as defined in the European legal regulation of the commission directive 1999/21/EC of 25 March 1999,¹ independent of the route of application. It includes oral nutritional supplements (ONS) as well as tube feeding via nasogastric, nasoenteral or percutaneous tubes. This definition differs from definitions used in many other publications where “EN” is rather used for tube feeding only regardless if blenderized food or specific industrial products are used. This decision was based on the fact that many studies

dealing with EN report on both ONS and tube feeding. Furthermore, prescription and reimbursement of EN is in many countries dependent of the use of industrial products rather than the route of application. EN is part of a qualified nutritional regimen in the in- and outpatient setting, and usually one of the tasks of professionals with special training in EN or the nutritional support team.”

ADVANTAGES OF ENTERAL NUTRITION

1. Less expensive, easier to administer, safe & more physiological for the patient[29].
2. Fewer complications compared to either types of nutrition
3. Maintains the histologic structure physiological viability of the gut.
4. It helps to maintain the immune system & the nutritional - metabolic axis.
5. It prevents bacterial translocation

6. Simpler system (easier for care giver or self- administration)
7. It maintains the hormonal balance, & stimulates the epithelial growth & regeneration.

General Indications

1. Patients who can not eat.
2. Patients who will not eat.
3. Patients who should not eat.
4. Patients who can't eat enough & where there is functional gastro intestinal tract.

Access Routes of Enteral Nutrition

1. Oral
2. Nasogastric tubes
3. Nasoenteric tubes (Nasoduodenal, Nasojejunal)
4. Cervical pharyngostomy
5. Gastrostomy
- Percutaneous endoscopic gastrostomy (PEG)

-Fluoroscopic gastrostomy

-Laparoscopic gastrostomy

-Surgical gastrostomy.

6. Jejunostomy

-Percutaneous endoscopic jejunostomy (PEJ)

-Laparoscopic Jejunostomy

-Surgical Jejunostomy

ACCESS TECHNIQUES

Naso gastric tubes (Ryle's Tube)

For Enteral nutrition stomach is the preferred organ as it allows use of more solid feeds, hypertonic feeds and higher feed volume. Ryle's tube feeds are the most commonly used for giving enteral nutrition.

Insertion:

1. Explain the procedure to the patient
2. Lubricate the tube externally with gel (or) water & internally with water if guide wire is present. Check guide wire moves freely.

3. Check nasal patency and lignocaine is sprayed through that nostril..

5. Sit the patient upright at hand level, slide the tube gently backwards.(Sniffing position)

6.Patient is given sips of water and as he swallows the tube is gently advanced..

7.Repeat the water swallow or advance until the preset mark on the tube reaches nostril.

If difficulty in passing the tube, ask the pt to tilt the head forwards (or) turn it to one side. Once in place remove any guide wire & secure. Check position of the tube before use. Document tube insertion in patients rates.

VERIFICATION OF TUBE PLACENENT :

Primary confirmation - Radiography

Secondary confirmation - Mark tube at exit site

REMOVAL OF NASOGASTRIC TUBE :

Before removing the Ryle's tube is flushed with 20ml air to empty the contents.

INDICATIONS :

Decompression of stomach & intestine.

DIAGNOSTIC :

Aspiration : Drugs, toxins.

Measurement : Gastric secretion, volume, pH

To procure specimens :of mycobacterium/ H. Pylori

THERAPEUTIC :

-Lavage & evaluation of gastric contents in upper GI bleeding
(or) toxin ingestion

-Sub acute intestinal obstruction/complete.

-Gastric dilatation, perioperative gastric damage, reduction
of risk for aspiration.

CONTRAINDICATIONS :

Nasopharyngeal obstruction, varices, coagulopathy,
thrombocytopenia, cranio facial injury. Recent foregut surgery.

COMPLICATIONS:

Associated in up to 15% hospitalized patients

Aspiration pneumonia is most common.

Emesis, gagging, epistaxis, sinusitis, alar pressure necrosis, odynophagia, nasopharyngitis, otitis.

Less common are esophageal strictures, perforation, laryngeal injuries, pulmonary complications.

Smaller tubes - occlusion.

TO AVOID COMPLICATIONS:-

- Proper placement
- Maintenance of up position.
- Proper insertion technique.
- Assessment of placement

PREVENTING TUBE OCCLUSION

Occlusion is generally due to the coagulation of protein based formula when they come in contact with acid or drugs.:

It is prevented by routine flushing with water after tube feeds and once every four hours.

GASTROSTOMY :

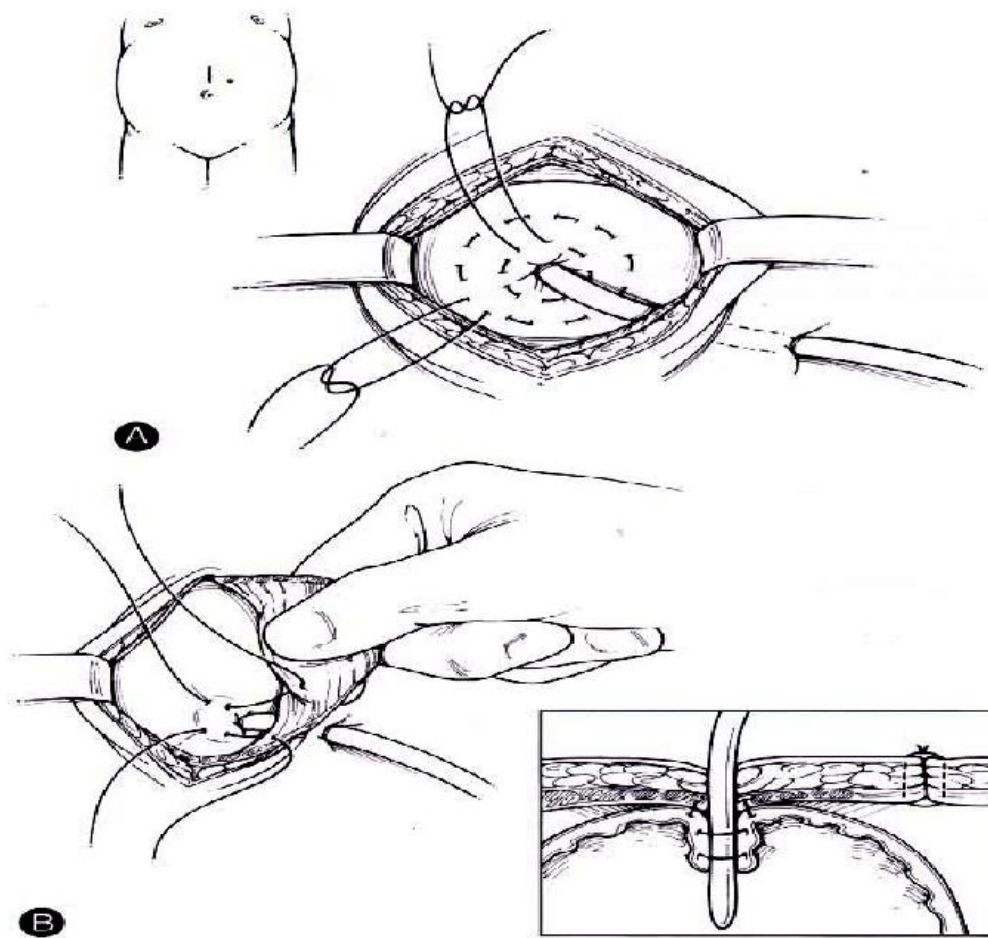
Intubation of the stomach (exclusive of the nasogastric route) results in planned gastrocutaneous fistula.

ADVANTAGES : Low leak rate, less cost, ease of placement, placed adjunctly with Gastro intestinal surgery. Spontaneous closure when removed.

DISADVANTAGES : Inadvertent tube removal results in rapid & premature loss of enteral access, risk of aspiration, stoma care needed, potential skin excoriation.

INDICATIONS : Head & Neck cancer. Cerebrovascular accident, trauma, respiratory failure. Prolonged intubation .

CONTRAINDICATIONS: Gastro esophageal reflux disease, gastroparesis, gastric outlet obstruction, pancreatitis, recent foregut surgery.



I) OPEN GASTROSTOMY : STAMM METHOD :

1. Gold standard for transabdominal gastric access.
2. Requires small laparotomy. Stomach is accessed via a small upper midline incision. Omentum & transverse colon identified & retracted inferiorly.
3. A relatively avascular site is chosen along the anterior wall of stomach, away from antrum & pylorus. The exit site should be in left upper quadrant.
4. A large bore (22-24f) tube often with a balloon (or) mushroom tip is placed through the abdominal wall through separate stab incision.
5. One (or) two purse string sutures are placed in seromuscular layer of anterior wall of stomach.
6. Create a gastrostomy in the middle of purse string suture.
7. Insertion of the tube done.
8. The balloon is inflated and the purse string sutures tied securely, anterior wall of the stomach affixed to abdominal wall entry site & tube secured to skin.

II) PERCUTANEOUS ENDOSCOPIC GASTROSTOMY

Indications :

- Patients requiring feeding for longer time.
- Dysphagia secondary to oropharyngeal cancer
- Neurologic event precluding swallowing. (CVA, multiplesclerosis).
- Tracheo esophageal fistula.

Contraindications:

- Coagulation disorder
- Marked esophageal obstruction
- Massive ascites
- Obstruction & prevdo obstruction
- Peritoneal dialysis/ metastases
- Respiratory distress

INTRODUCTION :

- PEG was introduced in 1980's by Gauderer & Ponsky
- Functional upper GI tract & prolonged enteral feeding are essential requirements for PEG placements.
- Permits feeding distally in the jejunum with gastric decompression.
- Well established & safe with minimum anaesthesia & complications.
- Currently method of choice for gastric intubation for nutritional support.

Technique of Percutaneous Endoscopic Gastrostomy:

This is generally done under iv sedation.

1. PULL THROUGH TECHNIQUE :

This is done by an endoscopist & his assistant.

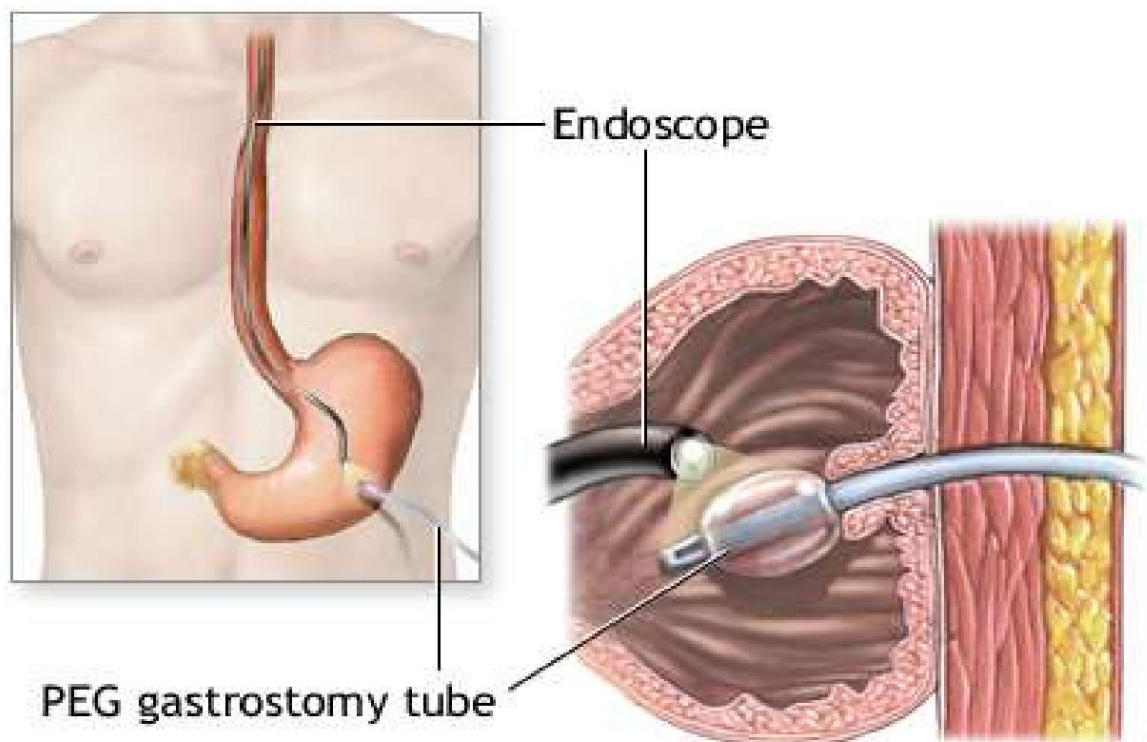
Endoscope is passed, patency of the gastric outlet is confirmed. Now indentation is given on the anterior wall of the stomach by the endoscopist.

Now the assistant introduces under local anesthesia a needle canula following the light into the anterior wall of the stomach. Silk thread is introduced through the cannula into the

stomach lumen, which is grasped and pulled out through the mouth through the snare.

Now the tapered end of the PEG tube is glided over the silk and silk thread is pulled through the anterior abdominal wall along with the PEG tube till the tapered end pierces out of the wall.

Now it is sinally pulled till the inner cup snugly fits the anterior wall of the stomach and tube is fixed to the exterior.



2. PUSH TECHNIQUE :

“Here a soft guide wire is passed through the needle catheter into stomach lumen which is pulled out of patients mouth using a snare. Now tension is applied at both ends of the wire while the tapered end of the gastrostomy tube is passed over it & pushed down into the stomach till it comes out of anterior abdominal wall. Then the tube is fixed to the abdominal wall.”

3. INTRODUCER TECHNIQUE:

1”.A split sheath introducer is passed over a J- tipped guide wire inserted into stomach lumen through a needle catheter.

2. The guide wire and the introducer are removed and a 14 french foley catheter is fed through the split sheath, which is ultimately peeled away.”

Complications:

Procedure related early (within 14 days) or late (after 14 days).

-Minor complication: Tube dislodgement, wound infection, fever.

-Major complications : peritoneal leakage with peritonitis, necrotizing fascitis of anterior abdominal wall, gastric haemorrhage, Perforation of stomach & colon.

LAPAROSCOPIC GASTROSTOMY[52]:

General anaesthesia & pneumoperitoneum is required

1. Approximation of the stomach to the abdominal wall is accomplished with T- fasteners placed percutaneously.

2. Four T- fasteners placed around the respective gastrostomy site.

3. A gastrostomy tube is then placed percutaneously through the center of T- fasteners into gastric lumen.

Stomach can be affixed to abdominal wall using sutures also & further held in place with an intraluminal balloon

FLUOROSCOPIC GASTROSTOMY:

Retrograde fluoroscopic -percutaneous technique used.

Fluoroscopic visualization of a needle puncture of stomach.

Creation of a tract over the guide wire done & tube fixed & anchored.

JEJUNOSTOMY :

Indications :

Recent surgery, Gastric outlet obstruction, gastroparesis, pancreatitis, fistula, esophageal reflux, high risk of aspiration.

Contra indications:

Short bowel syndrome, distal obstruction, inability to provide continuous infusion.

LAPROSCOPIC JEJUNOSTOMY :

`T' fasteners placed into antimesenteric border of small bowel under direct laproscopic visualization.

An introducer with a peel away sheath is placed into the jejunum through abdominal wall.

The `T' fasteners are cut at skin level,10-14 days later.

OPEN (WITZEL) JEJUNOSTOMY:

Laparotomy via a small upper Mid — line incision. Site 15-20cm distal to ligament of Treitz. Purse string suture placed on anti mesenteric border of jejunum. 14 F silastic tube is passed through the adjacent stab incision in soft upper quadrant.

Enterotomy is created through purse string.

Purse string suture is tightened, and a serosal tunnel is created proximally for approximately 3-5cm. Several sutures are used to affix the jejunum to the parietal peritoneum of the anterior abdominal wall at its exit site.

COMPLICATIONS OF ENTERAL NUTRITION :

I) GASTROINTESTINAL COMPLICATIONS:

1) NAUSEA & VOMITING:-

- 20% experience this complication
- It increases risk of aspiration
- Delayed gastric emptying is most common cause.
- If delayed gastric emptying is suspected. Consider reducing narcotic medications, reduce the rate of administration.

2) DIARRHOEA :

Most common in tube fed patients, occurring in 2% to 63% of patients. If clinically significant diarrhea develops during enteral tube feeding consider the following options:-

- Add fiber eg : psyllium.
- Consider an enteral formula with fiber.
- Change to formula .
- Use an antidiarrheal agent.

3) CONSTIPATION :-

Results from inactivity, decreased bowel motility, decreased fluid intake, impaction, lack of dietary fiber. Usually is improved through adequate hydration and use of fiber containing formulas, stool softeners, (or) bowel stimulants.

4) MALABSORPTION /MALDIGESTION:

Is defined as impaired absorption of one or more nutrients.

Clinical manifestations include unexplained weight loss, steatorrhea, diarrhea, anaemia, bone pain, glossitis, & edema.

II) MECHANICAL COMPLICATIONS :-

1) ASPIRATION:

- "Pulmonary aspiration is an extremely serious complication of enteral feeding & can be life threatening. symptoms include dyspnoea, Tachypnoea, wheezing rales, Tachycardia, agitation & cyanosis.

Risk factors for aspiration include:

- Diminished gag reflex
- Neurologic injury
- Incompetent lower esophageal sphincter

- Use of large bore feeding tubes.
- Large gastric residuals.

Presence of feeding tube itself may cause upper & lower air way complications, aggravation of esophageal varices, cellulitis, necrotizing fascitis, fistulas & wound infection.”

2) TUBE CLOGGING :

“ It is more likely due to intact protein products & viscous products. Prevention of clogging can be done by instilling warm water using slight manual pressure. If this fails, a pancreatic lipase & sodium bicarbonate solution may be instilled in order to digest the clog.”

III)METABOLIC COMPLICATIONS :

Problem	Cause	Treatment
Hyponatremia	Overhydration	Change formula, Restrict fluids
Hypernatremia	Inadequate fluids	Increase free water
Dehydration	Inadequate fluid intake	Increase free water, Evaluate causes of diarrhoea
Hyperglycaemia	Too many calories, lack of adequate insulin	Evaluate caloric intake. Adjust insulin
Hypokalemia	Refeeding syndrome, Diarrhoea	Replace, Evaluate causes of diarrhea
Hyperkalemia	Excess K intake, Renal insufficiency	Change formula

Refeeding Syndrome[20]

“Refeeding syndrome is a potentially lethal condition that can occur with rapid and excessive feeding of patients with severe underlying malnutrition due to starvation, alcoholism, delayed nutritional support, anorexia nervosa, or massive weight loss in obese patients. With refeeding, a shift in metabolism from fat to carbohydrate substrate stimulates insulin release, which results in the cellular uptake of electrolytes, particularly phosphate, magnesium, potassium, and calcium. However, severe hyperglycemia may result from blunted basal insulin secretion. The refeeding syndrome can be associated with enteral or parenteral refeeding, and symptoms from electrolyte abnormalities include cardiac arrhythmias, confusion, respiratory failure, and even death. To prevent the development of refeeding syndrome, underlying electrolyte and volume deficits should be corrected. Additionally, thiamine should be administered before the initiation of feeding. Caloric repletion should be instituted slowly, at 20 kcal/kg per day, and should gradually increase over the first week. Vital signs, fluid balance, and electrolytes should be closely monitored and any deficits corrected as they evolve.”

Enteral formulae[17]

“Any dietary food for special medical purposes designed for use in tube feeding or as an ONS. Enteral formulae can be

(1) nutritionally complete, when given in the recommended amount, to be used as a sole source of nutrition or as a supplement to the patient’s normal intake, or

(2) nutritionally incomplete, to be used as a supplement only and not as a sole source of nutrition.”

Oral nutritional supplements (ONS)

“Supplementary oral intake of dietary food for special medical purposes in addition to the normal food. ONS are usually liquid but they are also available in other forms like powder, dessert-style or bars. Synonyms used in literature: sip feeds.”

Nutritional support

“Nutritional support includes food fortification, ONS, tube feeding and parenteral nutrition as outlined in Fig. 1. It aims for increased intake of macro- and/or micronutrients. It is different from “special diets” which might be indicated in diseases like celiac disease.”

Standard formulae

“Standard formulae are enteral formulae with a composition, which reflects the reference values for macro- and micronutrients for a healthy population. Most standard formulae contain whole protein, lipid in the form of long-chain triglycerides (LCT), and fiber. However, non-fiber containing formulae with otherwise similar composition also exist.

Most standard formulae contain neither gluten nor lactose in clinically relevant amounts. The presence of gluten or lactose should clearly be mentioned on the label.”

Disease-specific formulae

“Disease-specific formulae include those with macro- and micronutrient compositions adapted into them.

Normal energy formulae provide 0.9–1.2kcal/ml, high energy formulae are anything above this, low energy formulae anything below.

High protein formulae contain 20% or more of total energy from protein.

Whole protein formulae contain intact proteins. Synonyms used in the literature: polymeric, high molecular weight or nutrient defined formulae

Peptide-based formulae contain protein predominantly in peptide form (2–50 amino acid chains). Synonyms used in the literature: oligomeric, low-molecular weight, chemically defined formulae.

Free amino acid formulae contain single amino acids as the protein source. Synonyms used in the literature: elemental, monomeric, low molecular weight, chemically defined formulae.

High lipid formulae contain more than 40% of total energy from lipids.

High MUFA formulae contain 20% or more of total energy from MUFA.

Normal diet of an individual as consumed at home/ in a restaurant/etc. or as offered by the catering system of a hospital. This includes special diets e.g. gluten-free, lactose-free diets.”

Fortified food

“Normal food enriched with specific nutrients, in particular with energy and/or proteins, minerals, vitamins, trace elements. Synonyms used in the literature: enriched food.”

DEFINITIONS

The following definitions are used in the guidelines.

Malnutrition

“Malnutrition is a state of nutrition in which a deficiency or excess (or imbalance) of energy, protein, and other nutrients causes measurable adverse effects on tissue/body form (body shape, size and composition) and function, and clinical outcome.”

Undernutrition

“Undernutrition is primarily used in the context of deficient energy or protein intake or absorption and is often described as protein energy malnutrition. It is frequently accompanied by multiple or single micronutrient and/or mineral deficiencies, although these may occur in the absence of macronutrient depletion and give rise to specific deficiency syndromes. Undernutrition may be due to a failure of food supply or intake, to deliberate fasting, or to disease and is characterized by weight loss and changes in body composition, which include loss of body fat, loss of lean mass (proportionately greater in disease compared to starvation alone) and a relative increase in extra- cellular fluid volume.”

Severe nutritional risk

“The term severe nutritional risk is used to describe the chances of a better or worse outcome from disease or surgery according to actual or potential

nutritional and metabolic status. Severe nutritional risk is defined as the presence of at least one of the following criteria:

weight loss 10–15% within 6 months,

BMI $\leq 18.5 \text{ kg/m}^2$, SGA Grade C or NRSX3,

serum albumin $< 30 \text{ g/l}$ (with no evidence of hepatic or renal dysfunction).”

Cachexia

“Cachexia is a term, which originates from the Greek words ‘kakos’, meaning ‘bad’ and ‘hexis’, meaning ‘condition’ (i.e. “bad condition”) and, in general, describes severe wasting from any cause including starvation and disease. Many clinicians use it as a qualitative term to describe the patient’s appearance of severe weight loss. Others have defined it quantitatively as a BMI $\leq 18.5 \text{ kg/m}^2$. More recently, it has also been used more specifically to describe wasting in life-threatening diseases such as cancer, AIDS, chronic obstructive pulmonary disease, and advanced organ failure where it is defined by a documented non-intentional weight loss of more than 6% in the previous 6 months, accompanied by catabolic conditions and resistance to increased substrate intake. In the current guidelines this latter definition of cachexia has been adopted.”

Wasting

“Wasting is used to characterise involuntary loss of body weight (i.e. muscle mass, “muscle wasting”) and decline of muscle strength. Wasting is not etiologically or pathologically different from under- nutrition but has been used customarily in certain contexts. The term “wasting syndrome” is established in the AIDS terminology as involuntary weight loss of more than 10% and/ either chronic diarrhoea (>1 month) and/or fever.”

Sarcopenia

“Sarcopenia describes a state of loss of muscle mass specifically occurring in bedridden, immobile or elderly patients.”

Nutritional screening

“Nutritional screening is a rapid and simple process conducted by admitting staff or community health care teams. The outcome of screening may lead to

(1) the patient is not at-risk of malnutrition, but may need to be re-screened at specified intervals, e.g. weekly during hospital stay,

(2) the patient is at-risk and a nutrition plan is worked out and implemented by the staff according to ordinary ward routines, or

(3) the patient is at-risk, but metabolic or functional problems prevent a standard plan being carried out or there is doubt as whether the patient is at-risk.

In any of these cases, referral should be made to an expert for assessment. Methods and application of nutritional screening have been described in a detailed ESPEN guideline (NRS).”

Nutritional assessment

“Nutritional assessment is a detailed examination of metabolic, nutritional or functional variables by an expert clinician, dietitian or nutrition nurse. It is a longer process than screening and it leads to an appropriate care plan considering indications, possible side effects, and, in some cases, special feeding techniques. It is based upon a full history,

clinical examination and, where appropriate, laboratory investigations including muscle function and bioelectrical impedance analyses (BIA). It will include the functional consequences of under- nutrition, such as muscle weakness, fatigue and depression. It includes gastrointestinal assessment, including dentition, swallowing, bowel function, etc. It necessitates an understanding of the interpretation of laboratory tests, e.g. plasma albumin, magnesium, phosphate, zinc, calcium and micro- nutrients. Subjective global assessment (SGA) is a widely used method of assessment.”

Ileus and Disorders of Intestinal Motility

Ileus and intestinal pseudo-obstruction designate clinical syndromes caused by impaired intestinal motility and are characterized by symptoms and signs of intestinal obstruction in the absence of a lesion-causing mechanical obstruction.

Ileus is a major cause of morbidity in hospitalized patients. Postoperative ileus is the most frequently implicated cause of delayed discharge following abdominal operations.

Ileus is a temporary motility disorder that is reversed with time as the inciting factor is corrected. In contrast, chronic intestinal pseudo-obstruction comprises a spectrum of specific disorders associated with irreversible intestinal dysmotility.

Pathophysiology

Numerous factors capable of impairing intestinal motility, and thus inciting ileus, have been described (Table 28-4). The most frequently encountered factors are abdominal operations, infection and inflammation, electrolyte abnormalities, and drugs.

Paralytic Ileus: Common Causes

Abdominal surgery	Sepsis	Intra-abdominal abscess	Peritonitis
Pneumonia	Hypokalemia	Hypermagnesemia	Hypothyroidism
Pancreatitis	Hypomagnesemia	Hyponatremia	Ureteral colic
Anticholinergics	Opiates	Phenothiazines	
Retroperitoneal hemorrhage	Myocardial infarction	Calcium channel blockers	
Spinal cord injury	Mesenteric ischemia	Tricyclic antidepressants	

Following most abdominal operations or injuries, the motility of the GI tract is transiently impaired. Among the proposed mechanisms responsible for this dysmotility are surgical stress-induced sympathetic reflexes, inflammatory response mediator release, and anesthetic/analgesic effects; each of which can inhibit intestinal motility. The return of normal motility generally follows a characteristic temporal sequence, with small intestinal motility returning to normal within the first 24 hours after laparotomy and gastric and colonic motility returning to normal by 48 hours and 3 to 5 days, respectively. Because small bowel motility is returned before colonic and gastric motility, listening for bowel sounds is not a reliable indicator that ileus has fully resolved. Functional evidence of coordinated GI motility in the form of passing flatus or bowel movement is a more useful indicator. Resolution of ileus may be delayed in the presence of other factors capable of inciting ileus such as the presence of intra-abdominal abscesses or electrolyte abnormalities.

Chronic intestinal pseudo-obstruction can be caused by a large number of specific abnormalities affecting intestinal smooth muscle, the myenteric plexus, or the extraintestinal nervous system (Table 28-5). Visceral myopathies constitute a group of diseases characterized by degeneration and fibrosis of the intestinal muscularis propria. Visceral neuropathies encompass a variety of degenerative disorders

of the myenteric and submucosal plexuses. Both sporadic and familial forms of visceral myopathies and neuropathies exist. Systemic disorders involving the smooth muscle, such as progressive systemic sclerosis and progressive muscular dystrophy, and neurologic diseases such as Parkinson's disease also can be complicated by chronic intestinal pseudo-obstruction. In addition, viral infections such as those associated with cytomegalovirus (CMV) and Epstein-Barr virus can cause intestinal pseudo-obstruction

Clinical Presentation

The clinical presentation of ileus resembles that of small bowel obstruction. Inability to tolerate liquids and solids by mouth, nausea, and lack of flatus or bowel movements are the most common symptoms. Vomiting and abdominal distention may occur. Bowel sounds are characteristically diminished or absent, in contrast to the hyperactive bowel sounds that usually accompany mechanical small bowel obstruction. The clinical manifestations of chronic intestinal pseudo-obstruction include variable degrees of nausea and vomiting and abdominal pain and distention.

Diagnosis

Routine postoperative ileus should be expected and requires no diagnostic evaluation. If ileus persists beyond 3 to 5 days postoperatively or occurs in the absence of abdominal surgery, diagnostic evaluation to detect specific underlying factors capable of inciting ileus and to rule out the presence of mechanical obstruction is warranted.

Patient medication lists should be reviewed for the presence of drugs, especially opiates, known to be associated with impaired intestinal motility. Measurement of serum electrolytes may demonstrate hypokalemia, hypocalcemia, hypomagnesemia, hypermagnesemia, or other electrolyte abnormalities commonly associated with ileus. Abdominal radiographs often are obtained, but the distinction between ileus and mechanical obstruction may be difficult based on this test alone. In the postoperative setting, CT scanning is the test of choice as it can demonstrate the presence of an intra-abdominal abscess or other evidence of peritoneal sepsis that may be causing ileus and can exclude the presence of complete mechanical obstruction.

The diagnosis of chronic pseudo-obstruction is suggested by clinical features and confirmed by radiographic and manometric studies. Diagnostic laparotomy or laparoscopy with full-thickness biopsy of the small intestine may be required to establish the specific underlying cause.

Therapy

The management of ileus consists of limiting oral intake and correcting the underlying inciting factor. If vomiting or abdominal distention are prominent, the stomach should be decompressed using a NG tube. Fluid and electrolytes should be administered intravenously until ileus resolves. If the duration of ileus is prolonged, TPN may be required.

Given the frequency of postoperative ileus and its financial impact, a large number of investigations have been conducted to define strategies to reduce its duration. Although often recommended, the use of early ambulation and routine NG intubation has not been demonstrated to be associated with earlier resolution of postoperative ileus. There is some evidence that early postoperative feeding protocols are generally well tolerated, reduce postoperative ileus, and can result

in a shorter hospital stay. The administration of NSAIDs such as ketorolac and concomitant reductions in opioid dosing have been shown to reduce the duration of ileus in most studies. Similarly, the use of perioperative thoracic epidural anesthesia/analgesia with regimens containing local anesthetics combined with limitation or elimination of systemically administered opioids have been shown to reduce duration of postoperative ileus, although they have not reduced the overall length of hospital stay. Interestingly, recent data have suggested that limiting intra and postoperative fluid administration can also result in reduction of postoperative ileus, and shortened hospital stay.

Measures to Reduce Postoperative Ileus

Intraoperative measures

- Minimize handling of the bowel
- Laparoscopic approach, if possible
- Avoid excessive intraoperative fluid administration

Postoperative measures

- Early enteral feeding
- Epidural anesthesia, if indicated
- Avoid excessive IV fluid administration
- Correct electrolyte abnormalities
- Consider opioid antagonists

Most other pharmacologic agents, including prokinetic agents, are associated with efficacy-toxicity profiles that are too unfavorable to warrant routine use. Recently, administration of alvimopan, a novel peripherally active μ -opioid receptor antagonist with limited oral absorption, has been shown to reduce duration of postoperative ileus, hospital stay, and rate of readmission rates.

REVIEW OF LITERATURE

“A study conducted in 24 patients who underwent elective surgery for esophageal carcinoma were randomized into immediate Enteral nutrition and parental nutrition group. This study showed beneficial effects on nutritional status, immunological competence, suppression of excessive inflammatory response, plasma nitrate and nitrite levels in the immediate Enteral nutrition group[2]. [Aiko S, Yoshizumi Y, Sugiura Y, Matsuyama T, Naito Y, Matsuzaki j et al, Beneficial effects of immediate enteral nutrition after esophageal cancer surgery. Surg Today 2001;31(11):971-8.]”

“.A prospective study trail in 212 patients who underwent pancreaticoduodenectomy were randomized to receive a standerd Enteral formula or parenteral nutrition. Patients receiving immunonutrition had a significant better recovery, decrease in rate of post operative complication($p=0.005$), mean length of hospital stay was shorter($p=<0.05$) This study concluded that post operative Enteral feeding may safely and effectively replace parental

nutrition in patients undergoing pancreaticoduodenectomy[3]. [Gianoiti L, Braga M, Gentilini O, Balzano G, Zerbi A, Dicarlo V. Artificial nutrition after pancreaticoduodenectomy 2000 Nov;21(1):59-65.]”

“In a study a total of 128 patients ,67 were randomized to a conventional return to diet group and 61 to free diet group. Results showed the complications are similar in both groups, free diet group tolerated normal diet well when compared to conventional group($p<0.001$). This study concluded that early resumption of oral intake does not diminish the duration of post operative ileus or lead to a significantly increased rate of nasogastric reinsertion, tolerance of oral diet is not influenced by gastrointestinal recovery, post operative management should include early resumption of diet [4]. [Han-Geurts J.M, Hop W.C.J, Kok N.F.M, Lim A, Brouwer K.J &Jeekel J. Randomized clinical trails of the impact of early enteral feeding on post operative leus and recovery BJS 2007;94:555-61.]”

“A metaanalysis of randomized controlled trail which included 11 studies with 837 patients showed early feeding reduces the risk of infection($p=0.036$),mean length of hospital stay ($p=0.001$),anastomotic dehiscence, wound infection, pneumonia, intraabdominal abscess and mortality.Finally concluded there is no clear advantage of keeping patients nil by mouth after elective gastrointestinal surgery,early feeding may be of benefit [5].[Lewis SJ,Egger M, Sylvester PA, Thomas S. Early Enteral versus "nil by Mouth" after gastrointestinal surgery: Systematic review and metaanalysis Of controlled trails BMJ 2001 Oct 6;323(7316):773-6.]”

“A study conducted on 104 successive patients who underwent colorectal surgery,89 patients started on oral diet out of which 65 patients tolerated early oral feeding. Univariate analysis showed that the use of volume expanders contributed to intolerance of oral feeding. In multivariate analysis, blood loss during the operation was the only factor contributing to failure of early post operative feeding. This study concluded early feeding is safe and feasible[6].[. Petrelli NJ, Cheng C, Driscoll D, Rodriguez-Bigas M A. Early post operative oral feeding after colectomy:an analysis of

factors that may predict failure. Ann Surg Oncol 2001Dec;8(10):786-800.]”

“A study conducted in 1716 patients after gastrectomy and surgeries for chronic duodenal obstruction showed that Enteral tube feeding stimulates motor, synthetic, and barrier function of small intestine, it also permits to improve immediate results of Stomach and duodenal surgeries and also reduces the cost of the treatment [7]. [Repin VN, Tkachenko IM, Gudkov OS, Repin MV Enteral tube feeding early after surgery on stomach and duodenum. Khirurgiia (Mosk) 2001(2):21-5]”

“A consultant physician D B A Silk had showed that early feeding may enhance wound healing and anastomotic strength particularly in malnourished patients. It is also associated with reduction in post operative complications and has beneficial effect on function of intestinal barrier in respect of permeability, bacterial translocation and subsequent development of septic complications [8]. [Silk D.B.A. Menzies Gow

N. Post operative starvation after gastrointestinal surgery early feeding is beneficial.BMJ 2001 Ooctober 6;323(7316):761-62]”

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“A study showed feeding gut early after surgery is safe and well tolerated and it should represented the first choice for nutritional support in these type of patients [9].[Braga M, Gianotti L, Gentilini O, Liotta S, Di Carlo V. Feeding the gut early after digestive surgery:results of nine year experience. Clin Nutr 2002 Feb;21(1):59-65].”

MATERIALS & METHODS

Source of data:

All patients undergoing elective laparotomies including upper gastrointestinal, hepatobiliary, colorectal and surgeries involving excessive bowel handling in the Department of General Surgery at Rajiv Gandhi Government General Hospital ,Chennai-600003 in the period of June 2013 to November 2013 are included in this study.

The study is a prospective study.

Inclusion criteria

1. Consent of the patient for the surgery as well as the study
2. Surgeries involving elective laparotomies
3. Patients above 12 years of age
4. Patient who had either oral or tube feeding will be included.

Exclusion criteria

1. Children less than 12years.
2. Emergency surgeries
3. Oesophageal surgeries.
4. Transplant surgeries.

100 patients undergoing elective laparotomies including upper gastrointestinal, hepatobiliary and colorectal surgeries are divided into two groups based on starting of enteral nutrition before or after 72 hours post operatively.

- Preoperatively adequate bowel preparation was done.
- For Gastric outlet obstruction ,stomach lavage with normal saline until clear aspirate was draining
- For colorectal surgeries ,Peglec Preparation was given orally preoperatively with soap water enema
- For small bowel surgeries, Peglec Preparation was given

- All patients were kept atleast 12 hours nil per oral preoperatively.
- I.V antibiotic to cover enteric flora was given at the time of induction.
- All surgeries were performed under epidural anaesthesia, general anaesthesia or spinal anaesthesia. Incision is made according to type of surgery performed.
- Post operative enteral feeding was started either orally or via ryles tube as per the discretion of the surgeon
- Plain water, medication, tender coconut water, lemonade, tea/coffee, were not considered as enteral nutrition.
- Intravenous fluids were given adequately during the immediate post operative period.
- If oral feeding was not tolerated(vomiting or abdominal distension) following intake, then patient is put on nil per mouth and Ryle's tube reinserted, Then orals are again started after 12 hours as tolerated by the patient.
- Time to acceptance of first oral/tube feeds(residual diet) was noted down.

- Patients were classified into Early Enteral Nutrition or Late Enteral Nutrition group based on whether time taken to start orals was less or more than 72 hours.
- Patients were slowly upgraded from low residue to semisolid to normal diet as per their tolerance.
- All cases were followed in the post operative period till they were discharged and later followed in the Out patient department

The following parameters were observed during the follow up in comparison between early and late feeding:-

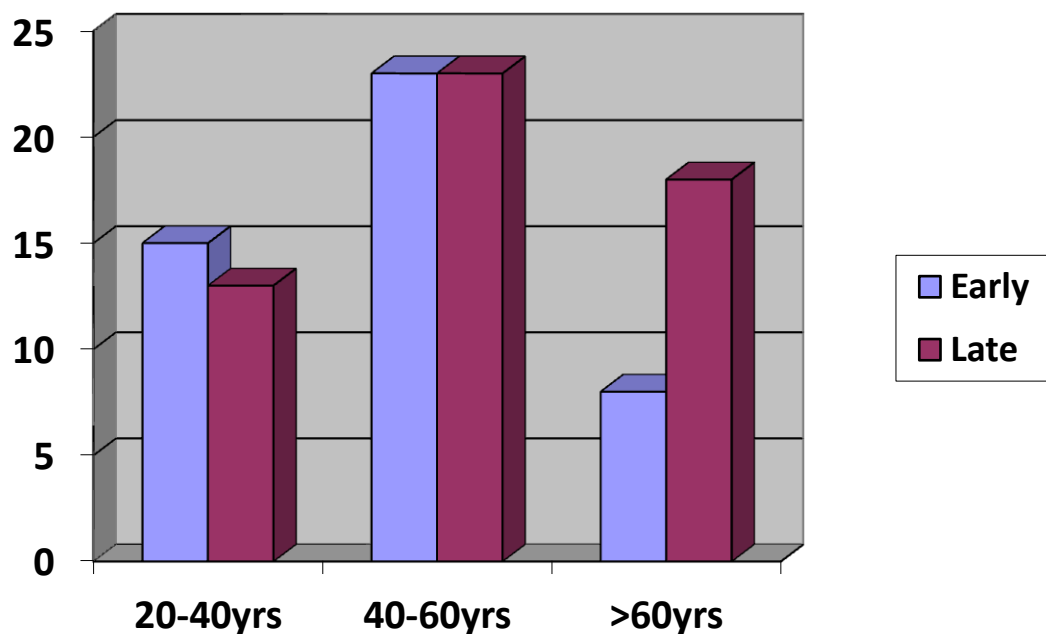
Length of hospital stay, Duration of surgery, Type of anaesthesia, Time of starting of orals and complications such as prolonged paralytic ileus, anastomotic leak, surgical site infection, tolerance of oral feeding, systemic complications including deep vein thrombosis, infections & mortality.

The results were analysed by Student t test to find significance of difference.

OBSERVATION AND RESULTS

A total of 100 patients were included in the study. 46 patients were included in the Early feeding group and 54 patients were included in the late feeding group. The following observations were made.

1.Age Distribution

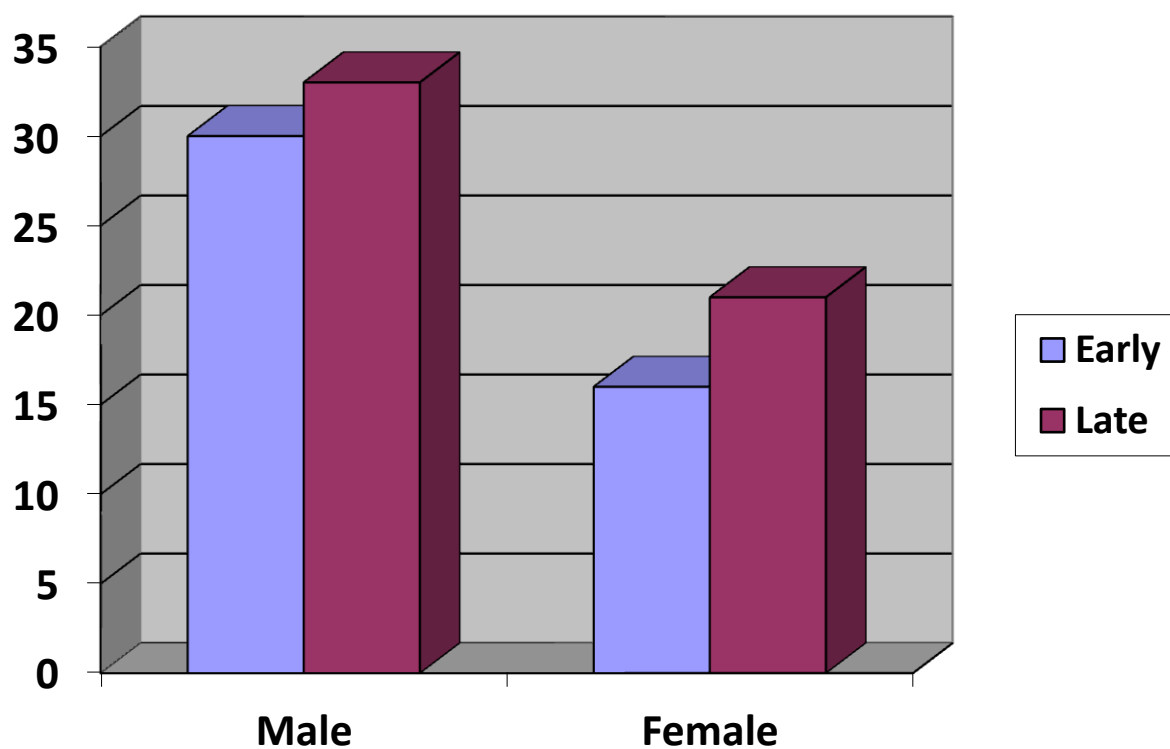


The mean age patients in the Early Enteral Nutrition Group was 46.65 years Standard deviation 16.37, Standard Error- 2.41 yrs.

The mean age of patients in the Late Enteral Nutrition group was 54.40yrs. Standard deviation 11.68, standard error - 1.59

Many of the patients were in >60 years age group in Late Nutrition Group(33.3%) as compared to Early Enteral Nutrition Group (17.5%).

2. Sex Distribution

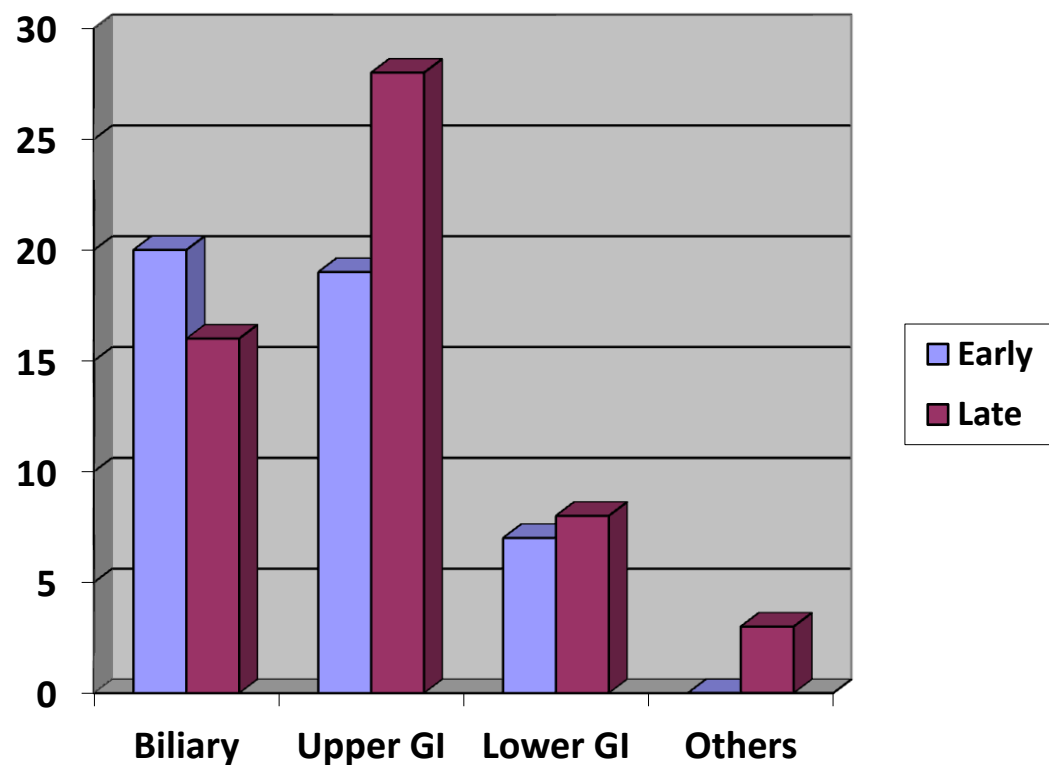


The sex preponderance was towards Male Sex in both the groups. The female to Male sex ratio in

Early Enteral Group was 53.33%.

Late Enteral Group was 63.63%

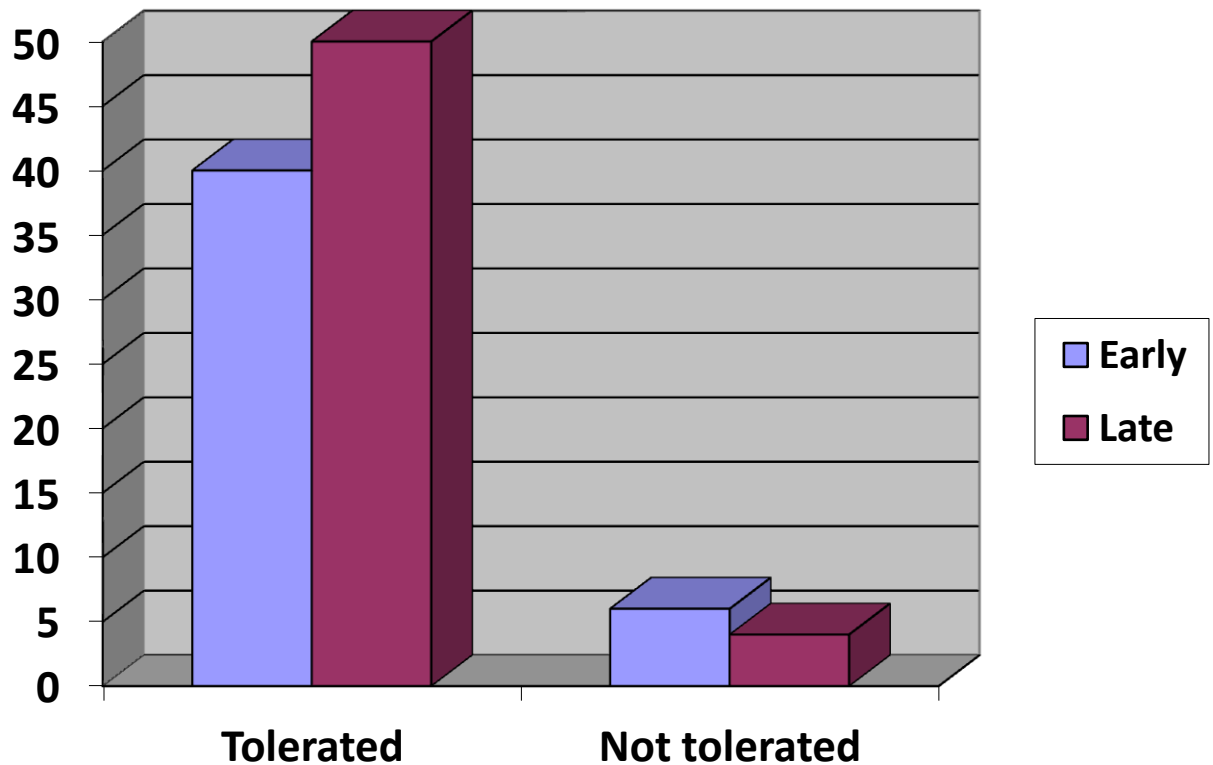
3. Type of Surgery



The commonest surgery in Early Enteral Nutrition Group was biliary tract surgery(44%) as compared to Late Enteral Nutrition Group where the predominant surgery was upper Gastrointestinal tract surgeries.(52%)

The other common surgeries done were Lower Gastrointestinal tract surgeries.

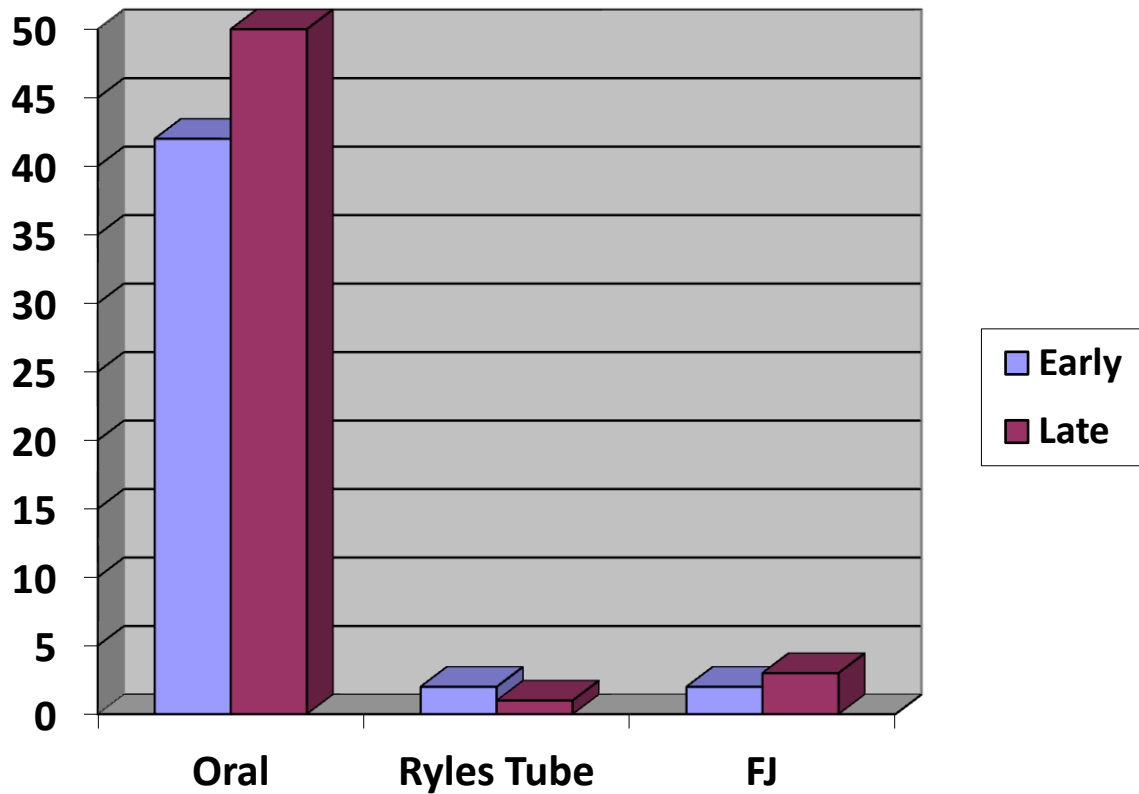
4.Feed Tolerance



Feed Tolerance was better in Late Enteral Nutrition Group as compared to Early Enteral Nutrition Group. 50/54 (92.59%) against 40/46(86.96%).

Non tolerance of enteral nutrition included abdominal distension following food intake necessitating cessation of oral feeds, vomiting, diarrhea after food intake.

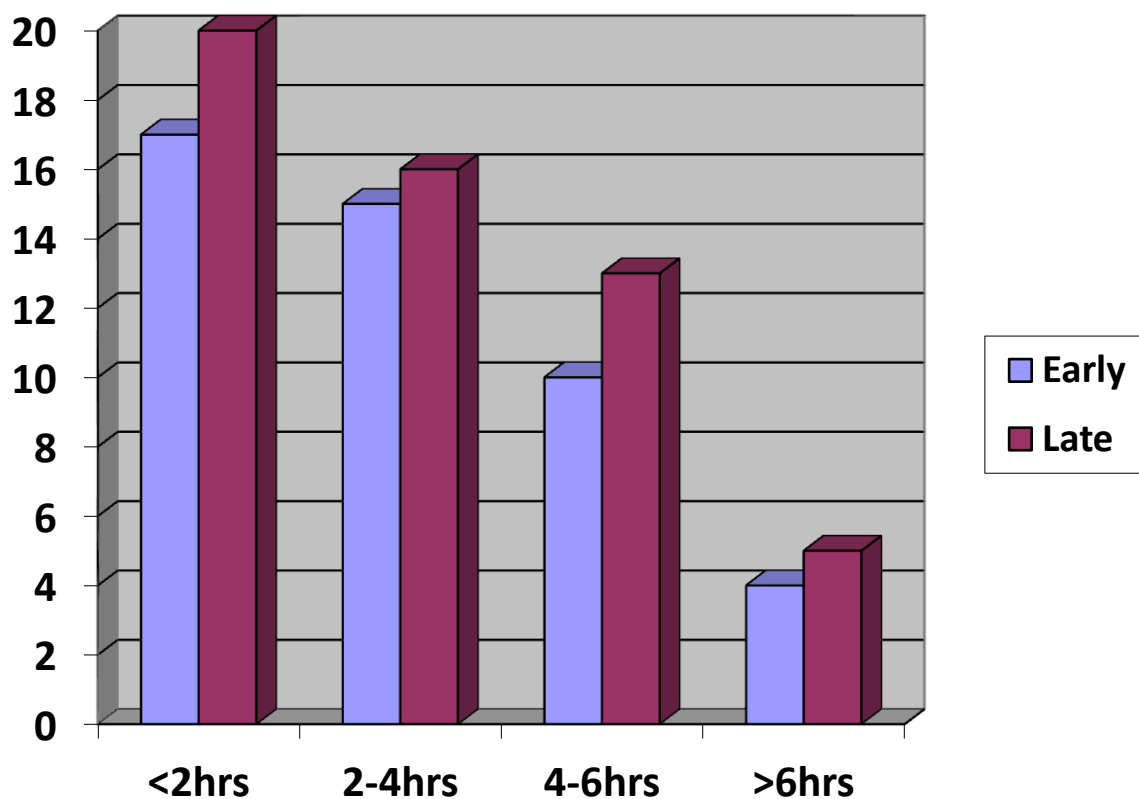
5.Mode of Enteral Nutrition



Most patients had enteral nutrition by oral route 91.3% in Early Enteral Nutrition Group versus 92.6% in Late Enteral Nutrition Group

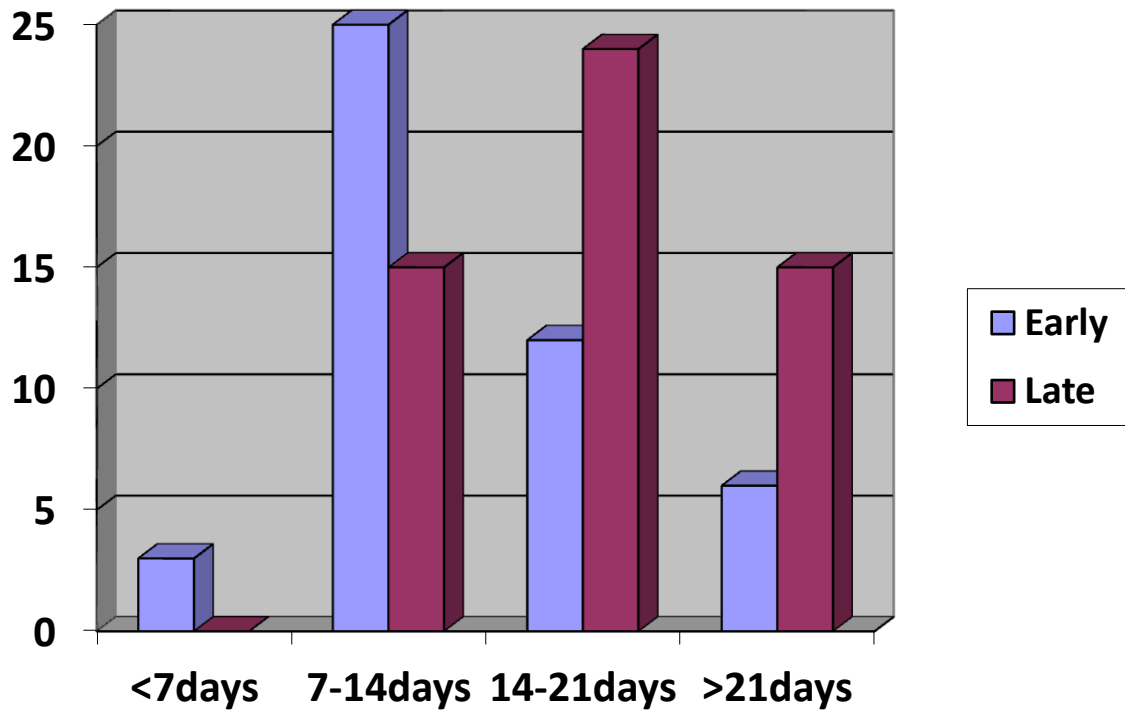
Other modes being Ryle's tube feeding and via feeding jejunostomy.

6.Duration of Surgery



The average duration of surgery in Early Enteral Nutrition Group was 180 mins with SD = 49.89, SE = 7.35 while it was 186 mins hours in Late Enteral Nutrition Goup. SD 67.43 , SE = 9.17. $p=0.59$. The difference was statistically insignificant. Most of the surgeries included in the study were within 6 hours.

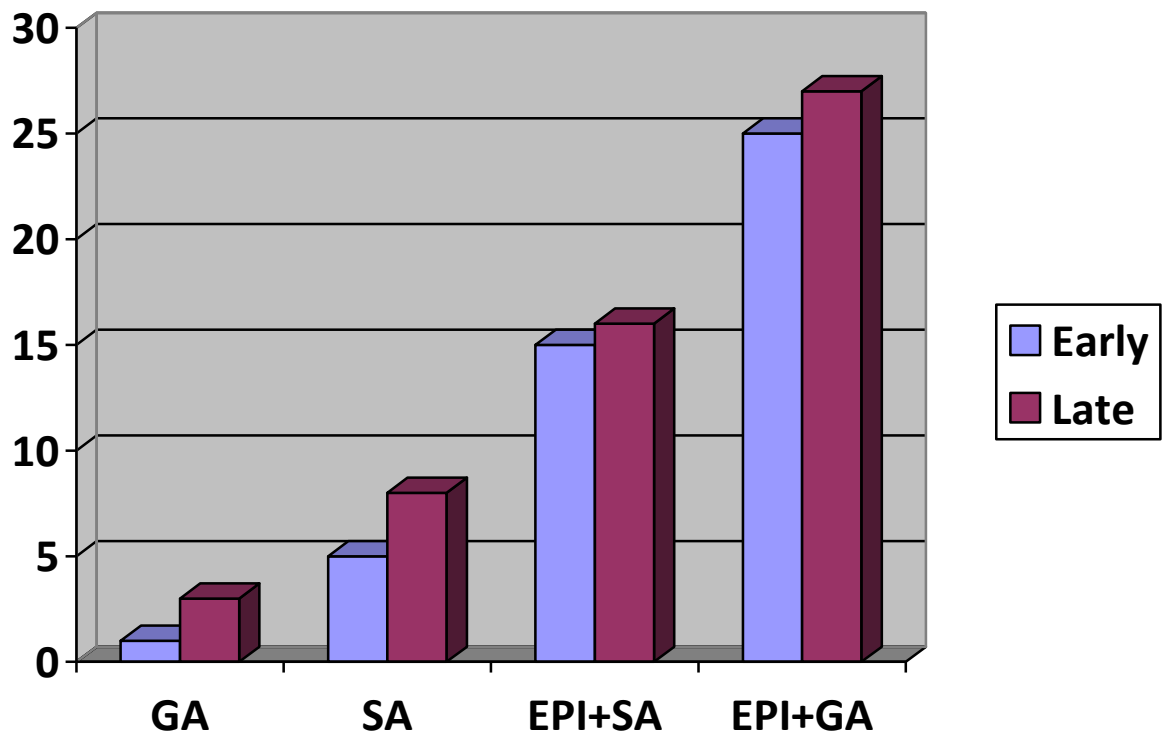
7. Length of Hospital Stay



The average duration of hospital stay in early enteral nutrition group was 14.46 days while it was 19.9 days in the Late Enteral Nutrition Group.

Hence it was 37.62 % increased hospital stay on an average for patients in the Late Enteral Nutrition Group.

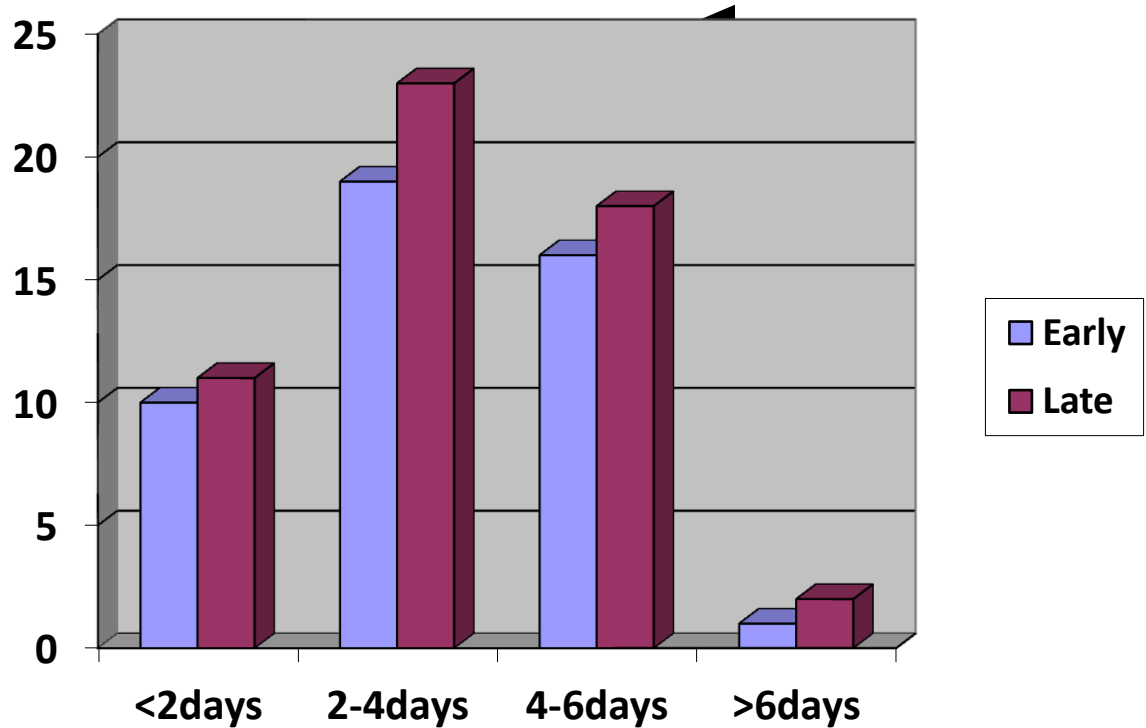
8. Type of Anesthesia



The Various forms of Anesthesia given to the patients undergoing the study were General Anesthesia(GA), Epidural Anesthesia(EPI), Spinal Anesthesia (SA) or in Combination.

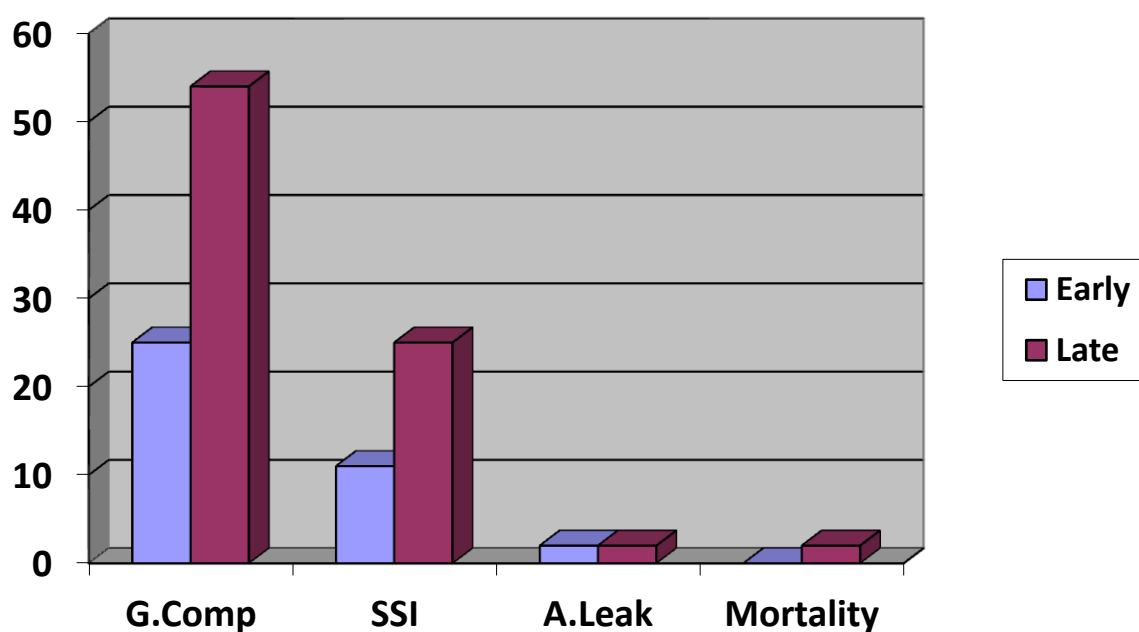
The most commonly employed anesthesia was General Anesthesia in combination with Epidural Anesthesia. (54.34%) in Early Nutrition Group as compared to (50%) in Late Nutrition Group.

9.Duration of Paralytic Ileus



The average duration for return of the bowel sounds was 49.48 hours in Early Enteral Group as compared to 54.18 hours in Late Enteral Nutrition Group which was not statistically significant.

10. Complications



General Complications(G. Comp) including pneumonia, Deep venous Thrombosis , urinary tract infections, cannula induced thrombophlebitis occurred as a total of 25 events in the Early Enteral Nutrition group as compared to 54 in Late Enteral group which was statistically significant.

Surgical Site Infections(SSI) occurred at 11 events in the Early group as against 25 events in the Late Group.

There was no statistically significant difference in the anastomotic leaks.

DISCUSSION

Feeding late after gastrointestinal surgery is the most common practice followed by many surgeons till date. There is no evidence suggestive that bowel rest and a period of starvation are beneficial for healing of wounds and anastomotic integrity. The advantages of early feeding is to reduce the length of hospital stay, early recovery, less incidence of complications such as wound infection. Discussion mainly emphasises on, firstly there does not seem to be a clear advantage of keeping patients nil by mouth after elective gastrointestinal resection, secondly in these patients early feeding may be beneficial.

The age distribution was towards the older age group in the Late Enteral Nutrition Group as compared to Early Enteral Nutrition Group. This may be a confounding factor as older age may again lead to increase in infections and complications rate.

The sex distribution was predominantly towards Male sex in both the groups.

The majority of the surgeries in the Early Enteral Nutrition Group was Biliary surgeries whereas it was Upper Gastrointestinal and small bowel surgeries that were more common in the Late Enteral Nutrition Group.

Epidural with General Anesthesia was the predominant mode of anesthesia used in both the groups.

The average duration of surgery in both the groups were almost similar.

Feed Tolerance was better in Late Enteral Nutrition Group as compared to Early Enteral Nutrition Group. 50/54 (92.59%) against 40/46(86.96%).

Non tolerance of enteral nutrition included abdominal distension following food intake necessitating cessation of oral feeds, vomiting, diarrhea after food intake. Starting orals in the setting of paralytic ileus may sometimes cause abdominal distension and vomiting which generally is self limiting. Oral feed may be momentarily stopped and started after sometime. Paralytic Ileus is not a contra indication for surgery and it is not necessary to wait till the appearance of bowel sounds to start the patient on oral feeds.

Starting on early orals prevents proliferation of pathogenic intestinal flora, transmigration across the intestinal mucosa and causing systemic infection.

Most patients had enteral nutrition by oral route in this study while other modalities including Ryle's tube feeding and Feeding Jejunostomy were used.

Most of the surgeries included in the study were within 6 hours.

The average duration of hospital stay in early enteral nutrition group was 14.46 days while it was 19.9 days in the Late Enteral Nutrition Group.

Hence it was 37.62 % increased hospital stay on an average for patients in the Late Enteral Nutrition Group.

This has various benefits including better bed turnover and economic benefits in a tertiary care hospital like our hospital.

General Complications including pneumonia, Deep venous Thrombosis , urinary tract infections, cannula induced thrombophlebitis occurred at a total of 25 events in the Early Enteral Nutrition group as compared to 54 in Late group.

Surgical Site Infections(SSI) occurred at 11 events in the Early group as against 25 events in the Late Group which were statistically significant.

There was no statistically significant difference in the anastomotic leak or mortality.

LIMITATIONS OF STUDY:

In this study age, sex, pre operative nutritional status, type of surgery, duration of surgery, type of anesthesia are not matched individually to compare the outcome, if these things are taken in to account then the comparison could be more accurate and the distinct advantages and disadvantages, can be assessed.

CONCLUSION

The final conclusions drawn from this study comparing early versus late enteral nutrition in the post operative patients undergoing elective laparotomies are as follows.

- Length of hospital stay is significantly decreased in early enteral feeding group.
- Paralytic ileus in both early and late feeding were the same.
- Wound infection was less in early feeding when compared to late feeding.
- No significant difference was noted with Anastomotic leak rate between two groups.
- Patients tolerance for oral feeding was better in late feeding.
- General complications not directly related to the surgery were more in Late feeding group.

This study has proven the advantage of starting early enteral feeding as compared to late enteral nutrition & clearly recorded that deferral of Enteral feeding was not beneficial.

BIBLIOGRAPHY

1. Lewis SJ, Egger M, Sylvester PA, Thomas S. Early Enteral feeding versus "nil by Mouth" after gastrointestinal surgery: systematic review and metaanalysis of Controlled trails.

BMJ 2001;323:773-6.

2. Aiko S, Yoshizumi Y, Sugiura Y, Matsuyama T, Naito Y, Matsuzaki j et Et al, Beneficial effects of immediate enteral nutrition after esophageal cancer surgery. Surg Today 2001;31(11):971-8.

3. Gianoiti L, Braga M, Gentilini 0, Balzano G, Zerbi A, Dicarlo V. Artificial nutrition after pancreaticoduodenectomy 2000 Nov;21(1):59-65.

4. Han-Geurts J.M, Hop W.C.J, Kok N.F.M, Lim A, Brouwer K.J&Jeekel J.

Randomized clinical trails of the impact of early enteral feeding on post operative leus and recovery

BJS 2007;94:555-61.

5. Lewis SJ, Egger M, Sylvester PA, Thomas S. Early Enteral versus "nil by Mouth" after gastrointestinal surgery:

Systematic review and metaanalysis Of controlled trails BMJ 2001 Oct 6;323(7316):773-6.

6. Petrelli NJ, Cheng C, Driscoll D, Rodriguez-Bigas M A. Early post operative oral feeding after colectomy:an analysis of factors that may predict failure.

Ann Surg Oncol 2001Dec;8(10):786-800.

7. Repin VN, Tkachenko IM, Gudkov OS, Repin MV Enteral tube feeding early after surgery on stomach and duodenum.

Khirurgiia (Mosk) 2001(2):21-5

8. Silk D.B.A. Menzies Gow N. Post operative starvation after gastrointestinal surgery early feeding is beneficial.

BMJ 2001 Ooctober 6;323(7316):761-62.

9. Braga M, Gianotti L, Gentilini O, Liotta S, Di Carlo V. Feeding the gut early after digestive surgery:results of nine year experience. Clin Nutr 2002 Feb;21(1):59-65.

10. Mercer D, Liu T: Open truncal vagotomy.

InOperativeTechniques in General Surgery 5:8-85, 2003.

11. McClave SA, Chang WK: Complications of Enteral access. Gastrointest Endosc 58:739,2003.

12. Davis RM: Complications of nasoenteric tubes. JAMA 254:54,1985.
13. Fox, K.A., Mularski, R.A., Sargati, M.R., et al.: Aspiration pneumonia following surgically placed feeding tubes. Am. J. Surg., 170:564, 1995.
14. Gomes GF, Pisani JC, Marcedo ED, Campos AC: Nasogastric feeding tube as a risk factor for Aspiration and aspiration pneumonia. Curr Opin Clin Nutr Metab Care 7:327,2003.
15. Marcuard SP, Stegall KL:Clearing obstructed feeding tubes. JPEN J Parenter Enteral Nutr 13:81,1989.
16. Mellinger, J.D., and Ponsky, J.L.: Percutaneous endoscopic gastrostomy: State ofthe art, 1998. Endoscopy, 30:126, 1998.
17. ESPEN Guidelines(www.espen.org/espenguidelines.html)
18. Ganong Review of Medical Physiology 24th eidtion
19. Skandalakis Surgical Anatomy 3rd Edition
20. Schwartz's Principles of Surgery 9th Edition

Abbreviations Used:

- **EN** - Enteral Nutrition
- **TV** - Truncal vagotomy
- **GJ** - Gastrojejunostomy
- **CBD**- Common bile duct
- **GOO** – Gastric outlet obstruction
- **CA** - Carcinoma
- **LAR** – Low Anterior Resection
- **LRI**- Lower Respiratory tract infection
- **UTI** – Urinary tract infection
- **SSI** – Surgical Site Infection
- **DVT** – Deep Vein Thrombosis
- **ST**- Superficial Thrombophlebitis
- **OJ** - Oesophagojejunostomy

PATIENT CONSENT FORM

STUDY TITLE:“COMPARATIVE STUDY BETWEEN POST OPERATIVE EARLY VS LATE ENTERAL NUTRITION IN PATIENTS UNDERGOING ELECTIVE LAPAROTOMIES IN A TERTIARY CARE HOSPITAL “.

STUDY CENTRE:Department of General surgery, Madras Medical College.

PARTICIPANT NAME:

AGE:

SEX:

I.P. NO :

I confirm that I have understood the purpose of interventional procedure for the above study. I have the opportunity to ask the question and all my questions and doubts have been answered to my satisfaction.

I have been explained about the possible complications that may occur during the interventional and interventional procedure. I understand that my participation in the study is voluntary and that I am free to withdraw at any time without giving any reason.

I understand that the investigator, regulatory authorities and the ethical committee will not need my permission to look at my health records both in respect to the current study and any further research that may be conducted in relation to it, even if I withdraw from the study. I understand that my identity will not be revealed in any information released to third parties or published, unless as required under the law. I agree not to restrict the use of any data or results that arise from the study.

I hereby consent to participate in this study of the **“COMPARATIVE STUDY BETWEEN POST OPERATIVE EARLY VS LATE ENTERAL NUTRITION IN PATIENTS UNDERGOING ELECTIVE LAPAROTOMIES IN A TERTIARY CARE HOSPITAL “**

Date:

signature / thumb impression of patient

Place:

Patient's name:

Signature of the Investigator:

Name of the investigator:

INFORMATION SHEET

We are conducting a study on

**“COMPARATIVE STUDY BETWEEN POST OPERATIVE EARLY VS LATE ENTERAL
NUTRITION IN PATIENTS UNDERGOING ELECTIVE LAPAROTOMIES IN A TERTIARY CARE
HOSPITAL “**

among patients attending Rajiv Gandhi Government General Hospital, Chennai and for that your information is valuable to us.

The purpose of this study is to find out the beneficial aspects including the reduction of morbidity, hospital stay & financial implications of early against late enteral nutrition in patients undergoing Gastro Intestinal surgeries.

We are selecting certain cases and if you are found eligible, we may be using your information which in any way do not affect your final report or management.

The privacy of the patients in the research will be maintained throughout the study. In the event of any publication or presentation resulting from the research, no personally identifiable information will be shared.

Taking part in this study is voluntary. You are free to decide whether to participate in this study or to withdraw at any time; your decision will not result in any loss of benefits to which you are otherwise entitled.

The results of the special study may be intimated to you at the end of the study period or during the study if anything is found abnormal which may aid in the management or treatment.

Signature of the Participant

Signature of the Investigator

Date

Place

Patient Proforma

Name :

Age :

Sex :

IP No. :

DOA:

DOS:

DOD:

Diagnosis :

Procedure Done:

Co-morbid illness :

Duration of surgery:

Type of anesthesia:

Enteral Feeding started at hours post operatively

Withdrawal or delay of enteral nutrition:/if yes reason:

Time taken for return of bowel sounds

Post Operative Complications:

Wound infections

Postoperative complications - systemic(including acute myocardial infarction,

Postoperative superficial/deep thrombosis, UTI or pneumonia)

Anastomotic leakages

Mortality

Length of hospital stay

ANNEXURE - MASTER CHART

S.NO	NAME	AGE/SEX	I.P.NO	DATE OF SURGERY	DIAGNOSIS	PROCEDURE	ANAESTHESIA	DURATION OF SURGERY(MIN)	MODE OF NUTRITION	TIME TO ORAL FEEDS IN HRS(GROUP)	TOLERANCE TO ORAL FEEDS	TIME TO RESUME BOWEL SOUNDS IN HRS	HOSPITAL STAY IN DAYS
1	VASUKI	26/F	51248	6/16/2013	CL	OC	GA	180	ORAL	24(E)	FAIR	36	7
2	VALLIYAMMAL	37/F	40980	6/17/2013	GB MASS	RAD CHOLE	GA	180	ORAL	72(L)	FAIR	48	14
3	SAMRAJ	59/M	48452	6/17/2013	CL	OC	GA	150	ORAL	30(E)	FAIR	36	18
4	KAMATCHI	33/F	47372	6/18/2013	CA STOMACH	SG &GJ&JJ	GA	300	RT	48(E)	FAIR	48	16
5	GOVINDHARAJ	60/M	52866	6/18/2013	CA STOMACH	SG &GJ&JJ	GA	300	ORAL	48(E)	FAIR	36	14
6	MURUGAN	70/M	49115	6/20/2013	CA STOMACH	SG &GJ&JJ	GA	360	ORAL	54(E)	FAIR	42	20
7	SUBRAMANIAN	48/M	48838	6/20/2013	GOO(ACID INGESTION)	GJ	GA	120	RT	78(L)	FAIR	48	21
8	AMUL	25/F	58163	6/20/2013	CA STOMACH	SG &GJ&JJ	GA	240	ORAL	36(E)	FAIR	48	21
9	LAKSHMI	40/F	44189	6/21/2013	CBD STRICTURE	CDJ	GA	195	ORAL	30(E)	FAIR	36	10
10	MAYAKANNAN	33/M	51532	6/22/2013	GOO	TVGJ	GA	150	ORAL	24(E)	NAUSEA/VOMITING	48	13
11	RAJALAKSHMI	65/F	48974	6/22/2013	CL	OC	EGA	180	ORAL	42(E)	FAIR	48	12
12	APPADURAIPIILLAI	76/M	57309	6/24/2013	CA STOMACH	SG &GJ&JJ	EGA	210	ORAL	48(E)	FAIR	54	16
13	ANTONY	40/M	55476	6/26/2013	PSEUDOCYST PANCREAS	CG	EGA	150	ORAL	36(E)	FAIR	48	13
14	BALARAMAN	41/M	57748	6/26/2013	ILEOSTOMY	RBC	ESA	150	ORAL	30(E)	FAIR	48	9
15	SUSEELA	75/F	57503	6/26/2013	CL	OC	GA	120	ORAL	24(E)	FAIR	36	10
16	SAVEETHA	18/F	58853	6/27/2013	MUCOCELE GB	OC	EGA	180	ORAL	48(E)	FAIR	48	10
17	SHARIFA	47/F	59937	7/5/2013	CL	OC	EGA	180	ORAL	24(E)	FAIR	36	11
18	SHANKAR	37/M	58688	7/6/2013	PSEUDOCYST PANCREAS	CG	GA	210	ORAL	48(E)	FAIR	54	11
19	MURALI	51/M	54995	7/7/2013	CCP	PUESTOWS	EGA	210	ORAL	78(L)	NAUSEA/VOMITING	96	22
20	ANJALACHI	50/F	62854	7/12/2013	CL&CDL	CL&CBDE	EGA	180	ORAL	72(L)	FAIR	54	12
21	SIVARAJ	60/M	120402	7/13/2013	GOO &CHOLELITHIASIS	TVGJ	EGA	180	ORAL	30(E)	NAUSEA/VOMITING	36	20
22	GERALD	53/M	57920	7/13/2013	CA RECTUM	APR	ESA	240	ORAL	72(L)	FAIR	60	24
23	KARTHICK	23/M	64710	7/15/2013	CL	OC	EGA	150	ORAL	24(E)	FAIR	42	6
24	THAVAMANI	32/F	61048	7/16/2013	GB POLYP	OC	EGA	180	ORAL	36(E)	FAIR	48	8
25	RAMAIAH	47/M	58633	7/17/2013	CL&CDL	CL&CBDE	EGA	150	ORAL	48(E)	FAIR	54	12
26	MUTHU	28/M	65445	7/18/2013	CL	OC	EGA	180	ORAL	24(E)	FAIR	48	9
27	DHAYALAN	55/M	63668	7/19/2013	CA STOMACH	GJ	GA	150	ORAL	24(E)	FAIR	42	10

28	CHINNAKULANTHAI	50/F	120307	7/19/2013	CA RT COLON	RT HEMICOLECTOMY	EGA	210	ORAL	60(E)	FAIR	84	21
29	KASTHURI	40/F	63563	7/20/2013	CL	OC	EGA	120	ORAL	78(L)	NAUSEA/VOMITING	96	20
30	CHANDRAN	57/M	64314	7/23/2013	CA STOMACH	SG &GJ&JJ	GA	210	ORAL	84(L)	FAIR	60	21
31	GAJENDRAN	60/M	64696	7/24/2013	CL	OC	EGA	180	ORAL	24(E)	FAIR	42	11
32	CHANDRAMOORTHY	60/M	68255	7/25/2013	CA RECTUM	LAR	ESA	180	ORAL	78(L)	FAIR	54	14
33	GOVINDARAJ	50/M	67015	7/26/2013	CL	OC	EGA	120	ORAL	30(E)	FAIR	48	12
34	SHAJAHAN	70/M	65030	7/29/2013	CL	OC	ESA	180	ORAL	36(E)	FAIR	54	10
35	DEVI	43/F	68634	7/30/2013	ILEOILEAL INTUSSUSCEPTION	RES&ANAS OF IL	EGA	90	ORAL	84(L)	FAIR	66	20
36	SUBAIYA	60/M	65024	8/2/2013	CL	OC	ESA	120	ORAL	84(L)	NAUSEA/VOMITING	36	19
37	VIMALA	40/F	69434	8/5/2013	D2 GROWTH	WHIPPLES PROC	EGA	300	FJ	90(L)	FAIR	72	25
38	GANESAN	49/M	71783	8/7/2013	CA STOMACH	SG &GJ&JJ	EGA	180	ORAL	78(L)	NAUSEA/VOMITING	72	18
39	KUMAR	38/M	59855	8/7/2013	CCP	PUESTOWS	EGA	180	ORAL	72(L)	FAIR	66	22
40	KARNAN	50/M	71796	8/8/2013	CL	OC	EGA	120	ORAL	28(E)	FAIR	48	15
41	NATHAMUNI	65/M	58074	8/10/2013	D2 GROWTH	WHIPPLES PROC	EGA	360	FJ	90(L)	FAIR	48	26
42	CHINAMMA	46/F	69654	8/12/2013	ANORECTAL GROWTH	APR	ESA	300	ORAL	78(L)	FAIR	54	21
43	RAJENDRAN	50/M	71081	8/14/2013	D3 GROWTH	WHIPPLES PROC	EGA	360	FJ	84(L)	NAUSEA/VOMITING	54	30
44	MUNIYAMMAL	50/F	74993	8/17/2013	SAIO	RES&ANAS OF IL	GA	120	ORAL	78(L)	FAIR	48	15
45	SELVAMANI	59/M	76753	8/19/2013	CL	OC	EGA	90	ORAL	72(L)	FAIR	48	12
46	ROSEMARY	35/F	62854	8/20/2013	GOO	TVGJ	EGA	180	ORAL	54(E)	FAIR	42	14
47	AMSAVALLI	67/F	77130	8/20/2013	CA RECTUM	LAR	ESA	180	ORAL	78(L)	FAIR	54	23
48	KUPPAMMAL	56/F	74767	8/20/2013	CL&CDL	CL&CBDE	ESA	120	ORAL	84(L)	FAIR	54	15
49	THANDAVARAYAN	75/M	75353	8/21/2013	CA STOMACH	SG &GJ&JJ	EGA	300	ORAL	78(L)	FAIR	48	19
50	SAVITHA	32/F	76505	8/21/2013	CL	OC	EGA	120	ORAL	36(E)	FAIR	48	15
51	ALAMELU	28/F	76616	8/21/2013	CL	OC	EGA	180	ORAL	24(E)	FAIR	42	16
52	RAJENDRA REDDY	61/M	72162	8/22/2013	CA STOMACH	TG &OJ	GA	180	ORAL	84(L)	NAUSEA/VOMITING	60	24
53	THOTTAMMA	68/F	78255	8/27/2013	ILEOSTOMY	ILEOSTOMY CLOSURE	SA	90	ORAL	72(L)	FAIR	48	16
54	VEERARAGHAVAN	40/M	76713	8/29/2013	RT PARAGANGLIOMA	RADICAL EXCISIO	EGA	240	ORAL	84(L)	FAIR	54	25
55	BHUVANESHWARI	30/F	81405	8/30/2013	CA RECTUM	APR	ESA	180	ORAL	60(E)	FAIR	54	21
56	SUJATHA	28/F	80836	9/5/2013	PERITONEAL CYST	RADICAL EXCISIO	EGA	240	ORAL	78(L)	FAIR	48	16
57	JINNA	58/M	78039	9/5/2013	POST ILEOSTOMY STATUS	(IN ILEOSTOMY CLOSURE)	EGA	120	ORAL	78(L)	FAIR	48	14
58	GOPAL	50/M	81761	9/6/2013	CA STOMACH	SG &GJ&JJ	EGA	150	ORAL	84(L)	FAIR	54	22
59	RAJINI	60/F	82177	9/6/2013	CL	OC	EPI	120	ORAL	78(L)	FAIR	48	18
120	RAVICHANDRAN	45/M	72050	9/11/2013	CL	OC	EGA	180	ORAL	84(L)	FAIR	54	14

61	BALAPPAN	34/M	83695	9/11/2013	CA LT COLON	LEFT HEMICOLEC	EGA	180	ORAL	60(E)	FAIR	54	23
62	CHIDAMBARAM	70/M	84520	9/13/2013	CA STOMACH	SG &GJ&JJ	EGA	180	ORAL	72(L)	NAUSEA/VOMITING	48	30
63	KALIYAN	50/M	79259	9/16/2013	CA STOMACH	SG &GJ&JJ	EGA	165	ORAL	78(L)	FAIR	54	20
64	MALLIGA BEGUM	50/F	86503	9/17/2013	CA LT COLON	LT HEMICOLECT	GA	210	ORAL	84(L)	FAIR	60	20
65	CHELLADURAI	57/M	87013	9/17/2013	CA STOMACH	SG &GJ&JJ	GA	195	ORAL	72(L)	FAIR	48	19
66	VIJAYASARATHY	29/M	86933	9/23/2013	GOO	TVGJ	EGA	210	ORAL	60(E)	NAUSEA/VOMITING	48	17
67	PALANI	78/M	81331	9/25/2013	CA STOMACH	TG &OJ	EGA	150	ORAL	90(L)	NAUSEA/VOMITING	54	21
68	ELLAMMAL	70/F	83176	9/26/2013	GOO	SG &GJ&JJ	EGA	180	ORAL	72(L)	NAUSEA/VOMITING	48	20
69	VELU	37/M	90149	10/3/2013	POST ILEOSTOMY STATUS(C	ILEOSTOMY CLO'	ESA	105	ORAL	84(L)	FAIR	48	12
70	THOMAS	76/M	89315	10/3/2013	CA STOMACH	SG &GJ&JJ	GA	180	ORAL	72(L)	FAIR	54	20
71	JERALD	53/M	901201	10/5/2013	POST APR ILEOSTOMY	ILEOSTOMY CLO'	ESA	90	ORAL	84(L)	FAIR	48	13
72	MANI	55/M	90632	10/8/2013	CA STOMACH	SG &GJ&JJ	EGA	165	ORAL	72(L)	NAUSEA/VOMITING	48	20
73	KALIAMMAL	61/F	80797	10/8/2013	SIGMOID COLON CA	LT HEMICOLECT	GA	180	ORAL	60(E)	FAIR	54	24
74	CHANDRASEKAR	58/M	90015	10/9/2013	SIGMOID COLON CA	LEFT HEMICOLEC	EGA	240	ORAL	60(E)	FAIR	48	21
75	KARTHIKEYAN	42/M	94902	10/15/2013	CA STOMACH	SG &GJ&JJ	GA	165	ORAL	66(E)	FAIR	48	14
76	MEGANATHAN	52/M	91047	10/17/2013	GOO(PUD)	TVGJ	EGA	120	ORAL	54(E)	FAIR	42	16
77	RAVI	37/M	92288	10/17/2013	CA STOMACH	SG &GJ&JJ	GA	180	ORAL	84(L)	NAUSEA/VOMITING	66	18
78	SUSEELA	75/F	94446	10/21/2013	CL	OC	EGA	180	ORAL	72(L)	FAIR	48	16
79	RAMACHANDRAN	75/M	97535	10/24/2013	CA STOMACH	SG &GJ&JJ	EGA	240	ORAL	66(E)	FAIR	48	20
80	RUKKUMANI	70/F	97136	10/25/2013	PERIAMPULLARY CA	TRIPLE BYPASS	GA	195	FJ	90(L)	FAIR	54	19
81	GANESAN	46/M	90762	10/26/2013	SIGMOID COLON CA	LEFT HEMICOLEC	EGA	195	ORAL	90(L)	FAIR	120	18
82	MURALI	22/M	97920	10/26/2013	GOO	TVGJ	EGA	180	ORAL	48(E)	NAUSEA/VOMITING	48	14
83	DHANABAKKIYAM	55/F	94367	10/28/2013	D2 GROWTH	WHIPPLES PROC	EGA	300	ORAL	90(L)	FAIR	54	30
84	MUNIYAMMAL	65/F	98386	10/30/2013	LT ADRENAL MASS	LT ADRENALECT	GA	210	ORAL	84(L)	FAIR	54	28
85	PERIYATHAMBI	75/M	98431	10/30/2013	CL	OC	EGA	150	ORAL	36(E)	FAIR	48	21
86	BABU	32/M	100873	10/31/2013	CL&CDL	CL&CBDE	EGA	150	ORAL	72(L)	FAIR	54	7
87	POOVAMMAL	50/F	101130	11/4/2013	CA STOMACH	SG &GJ&JJ	GA	165	ORAL	84(L)	FAIR	54	22
88	SUBRAMANIAN	56/M	104081	11/11/2013	CA STOMACH	SG &GJ&JJ	GA	180	ORAL	78(L)	FAIR	48	24
89	JEGADEESAN	55/M	104217	11/13/2013	GOO	SG &GJ&JJ	EGA	180	ORAL	48(E)	VOMTING	54	14
90	NATARAJAN	55/M	102750	11/13/2013	CA STOMACH	SG &GJ&JJ	GA	180	ORAL	78(L)	FAIR	48	23
91	MOHAN	58/M	104204	11/14/2013	PERIAMPULLARY CA	WHIPPLES PROC	GA	360	ORAL	90(L)	FAIR	60	28
92	KANNAYAN	65/M	102492	11/22/2013	CA RT COLON	RT HEMICOLECT	EGA	240	ORAL	90(L)	FAIR	54	18
93	PARIMALA	28/F	108703	11/27/2013	CL	OC	GA	180	ORAL	54(E)	FAIR	48	14

94	MARAGATHAM	56/F	111156	11/27/2013	CL	OC	EGA	120	ORAL	66(E)	FAIR	48	12
95	MANI	54/M	110212	11/29/2013	MUCOCELE GB	OC	GA	120	ORAL	66(E)	FAIR	54	14
96	SUBRAMANI	58/M	107392	11/29/2013	STATUS TRANSVERSE COLO	COLOSTOMY CL	ESA	120	ORAL	60(E)	FAIR	48	17
97	SAKUNTHALA	60/F	111244	12/4/2013	CA STOMACH	SG &GJ&JJ	EGA	165	ORAL	78(L)	FAIR	54	21
98	KRISHNAN	50/M	112773	12/7/2013	CA STOMACH	SG &GJ&JJ	EGA	150	ORAL	90(L)	FAIR	54	20
99	GOVINDASAMY	62/M	111211	12/9/2013	GOO	TVGJ	EGA	135	ORAL	72(L)	FAIR	54	18
100	MURUGESAN	52/M	117861	12/12/2013	ILEOCECAL TB SUBACUTE IN	ILEOTRANSVERSI	EGA	150	ORAL	60(E)	FAIR	54	16